TRANSACTIONS

OF THE

THIRTIETH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

HELD AT

CHICAGO, ILLINOIS,

OCTOBER 19-24, 1925

The Thirty-first Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology will be held at Colorado Springs, Colo., September, 1926. The attention of the members is called to the ruling of the Council, that when a paper published in the Transactions is illustrated, the author must pay the total cost of all colored plates, and one-half of the cost of all other cuts. The Editor will furnish an estimate of the cost of such cuts and plates upon request. (*All papers, with illustrations, must be handed to the Secretary immediately after they are read.*)

Only those members who have paid their dues by March 1st of the year for which the Transactions are published will receive the Transactions. Those who pay after that date may obtain a copy upon payment of $5.00 additional.

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Members are requested to notify the Secretary of any error or omission in names, addresses or specialties in the list of members; also, of any change in address, before the time of the 1926 meeting.

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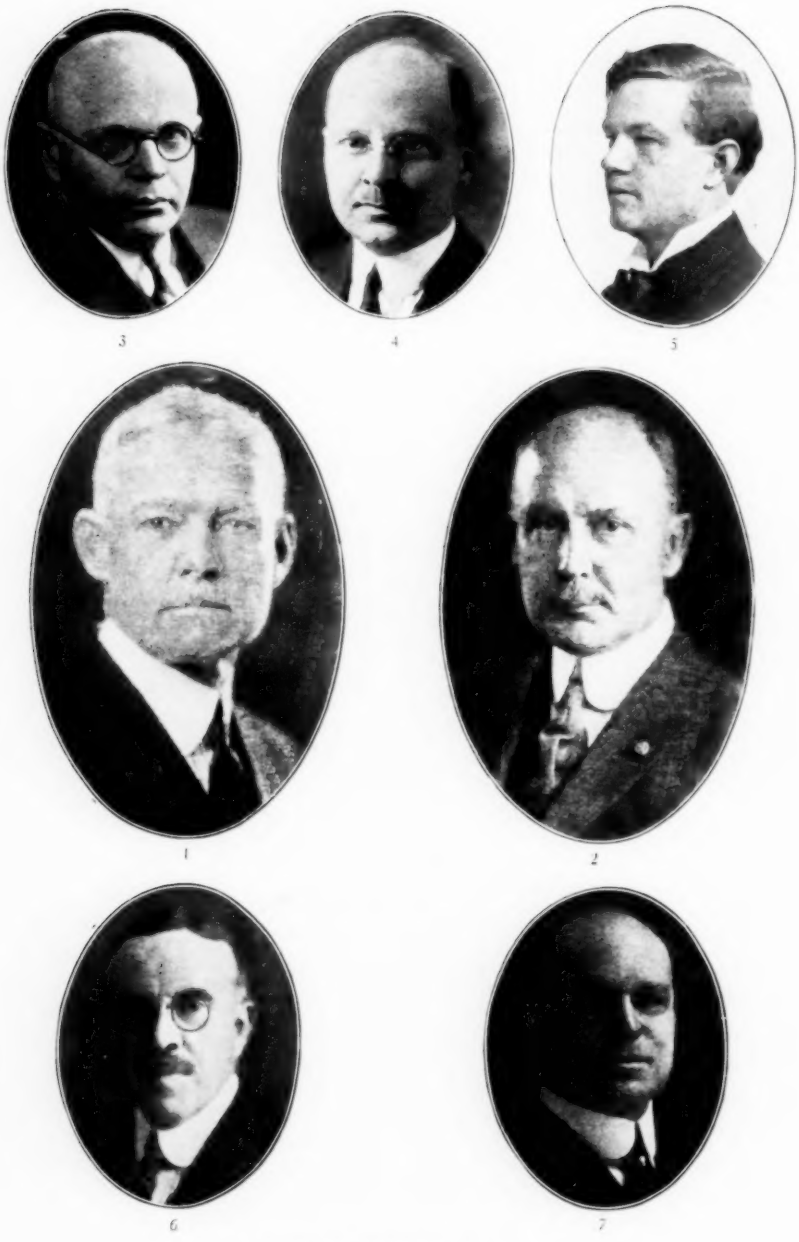

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TRANSACTIONS

OF THE

THIRTIETH ANNUAL MEETING

OF THE

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JOINT SESSIONS

PRESIDENT'S ADDRESS.

HORACE NEWHART, M.D.

MINNEAPOLIS, MINNESOTA.

Fellows of the Academy, Honored Guests, Ladies and Gentlemen:

By virtue of the authority with which you so graciously honored me a year ago, it is my great privilege to welcome you to this, the thirtieth annual meeting of the American Academy of Ophthalmology and Oto-Laryngology. On behalf of the Officers and Council, I would express the hope that every one here will feel the inspiration of the kindly interest and cordial good fellowship which has so characteristically dominated our meetings in the past, and has been largely responsible for the Academy's present scientific and numerical strength.

To those who are in our midst for the first time as recently elected Fellows or as candidates for Fellowship, I would extend a special welcome. The Academy has much to offer you through congenial contact and friendship and helpfulness, and in turn expects much of you in the way of endeavor and faithfulness in advancing its standards and in maintaining its place in American ophthalmology and otolaryngology.

It has long been the custom in societies such as this for the presiding officer at this time to present a learned scientific contribution based upon his personal investigations, or as an alternative, to unburden his mind of thoughts, the result of long and deep meditation upon some of the problems in which as a group we are particularly interested. The former function I have been pleased to delegate to our distinguished guests and to other contributors to the program. I have chosen to follow the second alternative.

It has been frequently said that our race has been able to achieve noteworthy progress only as men have learned how to cooperate better in whatever undertaking was before them. As members of a scientific body we have assumed the task of advancing the standards of one or more of the fields of

ophthalmology, rhinology, laryngology or otology as they are related to the clinical practice of medicine. Our attainments must vary greatly with individual capacity and opportunity. But few are blessed with the imagination and aptitude for productive research. But each one has in himself a large capacity for cooperative effort, a fact which has been largely overlooked because of the medical man's very marked tendency to individualism. By force of tradition and habit, the physician has been an extreme individualist—in the minds of the layman ultraconservative, noncommunicative, self contained and too often self sufficient. Through the exhibition of these very qualities which in the past have contributed in no small measure to his success, in these later years of widespread popular scientific knowledge, he has in some respects become noticeably alienated from the public, who are prone to misunderstand his aloofness and what they regard as his assumed superiority.

There is a great need today for a better understanding between the laity and the medical profession, to the end that the people may realize the significance of a scientific medical education and the service which the well trained medical man is prepared to render. The public should be taken more completely into our confidence, not only as to our achievements, but also as to the limitations of medical science, so that they may know by comparison how little can be done for them by the exponents of pseudomedical cults, whose commercial motives and base methods should be made known to them. The people need and are entitled to receive authentic information upon all lines of personal and public health.

The attainment of these ends calls for an effective cooperation among medical men; which means intelligent group action, beginning with a wisely directed publicity and a high minded spirit of service. With the objective a worthy one and the methods employed laudable, under wise guidance, the medical profession may expect from a more enlightened public a generous response and willing cooperation in its efforts to improve health conditions throughout our country. Each member of this Academy, after demonstrating his worth through the high quality of his service, by exercising his powers of leadership, can do large things for the advancement of his particular branch of medical science in his own community.

A problem which is today seriously confronting the ophthalmologist is involved in the rapid encroachment upon his special field by men who have had no medical training, and who are guided by no higher motives than commercialism. Backed by large funds for publicity, and with an aggressiveness which no group of professional men has yet manifested, they have successfully established creditable courses in physiologic optics and optometry in several of our leading universities, and even threaten ultimately to deprive the medical man of the right to undertake the correction of refractive errors by prescribing eye glasses, except as he has prepared himself according to their standards and shall be licensed by their boards of examiners. This is a problem which should receive the serious consideration of those who are concerned with the best interests of ophthalmology. The remedy is to be found in publicity, in better service on the part of those medical men doing eye work, and in a possible revision of the teaching of ophthalmology in our medical schools. (Ultimately it may be necessary to invoke legislation to give us special practice acts regulating the practice of ophthalmology as a special branch of medicine.)

A subject well worthy of a place upon our program would be a symposium or report based upon an investigation of the effects upon the vision of the consumption of alcoholic beverages since the passage of the Volstead act. An accurate statement of the real facts as established by the research of the ophthalmologists of our country would yield information of great value from a medical, sociologic and legal standpoint.

Conspicuous among the problems of medical science which as a group we have sadly failed to solve, is that of the prevention of deafness and the conservation of hearing. It is an unpleasant commentary upon our capacity to cope successfully with a situation, when it can be said without fear of denial that we have in our country 3,000,000 persons whose hearing acuity is so seriously impaired as to handicap them in their educational, economic and social contacts. The situation becomes all the more embarrassing, especially to the otologist, when we are forced to admit that the majority of these cases might have been prevented by relatively simple measures which should be at the command of every medical practitioner.

In comparison with what has been done under the leadership of the ophthalmologist for the conservation of vision, our

progress in any organized attempt at conserving the hearing, until very recently, has been almost negligible. Several well recognized factors have contributed to our failure to make a similar showing as regards the organ of hearing. We may mention among them:

(1) Inadequate training of the general practitioner in the diagnosis and treatment of the simpler diseases of the ear and of pathologic conditions leading to impairment of the ear.

(2) Indifference on the part of the general practitioner and on the part of the laity to ear disease, due largely to poor results attained in the treatment of these conditions.

(3) The fact that an individual may in many cases lose a considerable percentage of his hearing acuity without his knowledge, until the loss is irreparable when discovered.

(4) The fact that the organ of hearing is inaccessible for examination and treatment, and that its examination to detect a slight hearing loss, which would be indicative of beginning deterioration, has been difficult and calls for patience, time and a special environment affording quiet.

(5) Our limited knowledge of the etiology and pathology of many diseases of the ear.

During the past few years there has been a notable awakening of interest in the subject of the conservation of hearing. The public, recognizing the importance of preventative medicine in a general way, now rightly demand its application to the preservation of our special sense organs.

Organizations, composed largely of the deafened themselves, but including also interested otologists and public spirited citizens, have been formed in many of our larger cities, with the object of meeting the problems of the deafened. They are accomplishing much to help in the rehabilitation of those who have lost their hearing, and are active in providing for their economic, educational and social needs. These local associations have united in the formation of the American Federation of Organizations for the Hard of Hearing, which organization, through its Scientific Committee, is doing most creditable preliminary work in the prevention of deafness, which it recognizes as one of its most important functions. This committee, numbering among its members prominent physicists, otologists and educators, is seriously committed to the work it has undertaken and is determined to attain definite results.

This Academy, through your committee on the Hard of Hearing, is attempting to do its share in the solution of these problems. Several of the members of your committee are also members of similar committees of our other national otolaryngologic societies (the American Otological Society, the American Laryngological, Rhinological and Otological Society, and the Section on Laryngology, Otology and Rhinology of the American Medical Association), and are likewise members of the Scientific Committee of the American Federation. By this arrangement there is afforded the greatest facility for exchange of ideas and for well organized effort. At its last meeting the American Medical Association upon the motion of one of our own Fellows, Dr. Keiper, generously endorsed the work of the American Federation. Your chairman ventures the hope that the Academy will see fit to take a similar action at this meeting.

While much has already been accomplished by way of lessening the incidence of diseases of the ear leading to hearing loss, chiefly through better hygiene and the prevention of the acute exanthematous diseases of childhood, the problem as a whole has thus far been met only in a very small way.

By far the most advantageous point at which the problem of preventing deafness may be effectually attacked is in the public schools and in our educational institutions generally. Here we have grouped under more or less efficient medical supervision millions of our future citizens, at an age when measures calculated to conserve their special sense organs will prove most successful. The solution of our problem is to be found largely in regulations which will insure to each individual of school age a regular yearly examination of his hearing acuity (and at more frequent intervals in all such cases as demand it). These examinations would promptly reveal which pupils should receive attention.

Laws already exist in no less than twenty of our states providing for examination of the hearing acuity of school children, but as they have been administered in the past they have been of little service so far as they have been useful in detecting slight hearing loss. At the best, the average ear examination as carried out in our schools has been made with the object of determining which children are so badly incapacitated as to require special instruction. With few exceptions, when groups have been examined for scientific

purposes, as in Chicago and other cities when surveys have been made, these examinations have been without value for the purpose of detecting pupils with a slight hearing loss, among whom are many cases of potential deafness which will manifest itself later in life. Examinations sufficiently accurate to detect these cases are difficult of execution and entail the expenditure of much effort, time and means.

Today, as the result of the contributions of the physicist in the field of audiometry, we are much nearer the goal. The most outstanding advance in otology during the past decade is the development of audiometry, which marks the beginning of a new era in this branch of medicine. For this we are indebted to the investigations of Dean, Bunce, Knutsen, Krantz, Fowler, Jones and others, and lastly, Dr. Harvey Fletcher and his associates of the American Bell Telephone Laboratories, who have placed audiometry on a basis which makes it practically available in a clinical way.

One type of audiometer has lately been perfected, by means of which reasonably accurate tests may be simultaneously made upon groups of from eight to forty individuals. Herein lies in a large measure the solution of the problem of the early detection of impaired hearing acuity. The otologist, the general practitioner and the educator alike are quick to see the great advantage of this wholesale method of procedure. Those with subnormal hearing can at once be easily segregated from the normal, and the need of treatment be made plain to those responsible for the health of the child. Upon the basis of preliminary work along these lines which some of us have already carried out, I feel justified in predicting the early establishment of a diagnostic ear clinic as a part of our public school system. Such clinics are already in successful operation in a few of our larger cities. Even in our smallest communities, by the use of the audiometer it will be possible to make accurate hearing tests of all school children, for in case of necessity, the tests can be successfully carried out by the school nurse or the teacher.

The next logical step in the solution of the problem of the prevention of deafness must be a propaganda of education among the public, which will result in the enactment in all of our states of uniform laws providing for the adequate testing of the ears of all children of school age. Long before this is accomplished, the methods which have been here outlined, be-

cause of their simplicity and effectiveness, will probably be voluntarily put in force in the schools of our larger and more progressive communities. The results within a few years must prove of inestimable value in saving thousands of individuals from the embarrassment and handicap of an unnecessary hearing loss.

I can not close without once more expressing to you my deep appreciation and gratitude for the honor you conferred upon me by making me your chief executive officer during the past year. My own small efforts in behalf of the Academy would have been futile without the constant help of our tireless and incomparably faithful secretary, Dr. Peter. And in all things I have been conscious of the strong support of your officers and Council, whose close fellowship and friendship has been a joy, and whose unselfish and never failing devotion to the Academy has been a constant inspiration.

SYMPTOMATOLOGY AND DIAGNOSIS OF EXPANDING LESIONS OF THE BRAIN, WITH SPECIAL REFERENCE TO DISTURBANCES OF VISION, HEARING, TASTE, SMELL AND SPEECH.

FOSTER KENNEDY, M.D.

NEW YORK CITY.

Mr. President and Gentlemen: I would say first a word of thanks to you for the honor you have paid me, in asking me to come to this great city to address you on problems which press for solution by men of your trades and of mine. It seems fitting somehow that I should come to Chicago—though unmerited that I should come on so excellent an errand—for this city was the first part of America with which as a small boy I made even indirect contact. A relative of mine who visited the World's Fair in 1893 brought back to me a book from here about Columbus, and my childish pleasure at that, my first gift from America, is now distanced by that afforded by the gift of your courtesy to me.

The classical triad, headache, vomiting and papilledema, are signs of increased intracranial tension, and in the absence of cardiorenal disease, are presumptive evidence of an intracranial expanding lesion. They are by no means always present. Perversion and deterioration of function of progressive character may be so exact, that the observer can be sure of the presence of tumor or abscess formation before pressure conditions are such as to display general signals.

It is, indeed, admirable when we can anticipate such signals, but experience is needed for the correct interpretation of small progressive signs. At one time, we thought a pathognomonic sign of imminent intracranial tension lay in the interlacing or reversal of the color fields; this hope has not been realized by experience. Attacks of dizziness occur often early in the history of cases of expanding lesions of the brain, due to irritation of the labyrinths or other parts of the cerebellar mechanisms; such vertigo may be dependent upon or synchronize with transient diplopia. Before such an audience as

this, it would be improper for me to emphasize the physiologic reasons causing an inverse ratio between the degree of diplopia and the degree of vertigo produced by the diplopia, but a word is in place, I think, for Cushing's observation accounting for transient sixth nerve palsies. Cushing found that often these nerves are overlaid by branches of the basilar artery, and that gross arterial indentations can be seen across nerves so disadvantageously placed; probably this anatomic situation explains some apparently anomalous cases of sixth nerve paralysis in arteriosclerotic persons.

A good deal of controversy has taken place regarding the mechanism of choked disc, so often improperly called optic neuritis; I hope that the oculists have been able to accept as a true theory, that as a result of an expanding intracranial lesion, the contents of the cerebral pond are forced to dilate the ventricles, to drag and stretch the cerebral membranes, to push the inverted cone of the medulla into the bony foraminal ring, inducing nausea and vomiting and a slow vagal pulse, and finally to force cerebrospinal fluid out of the great cerebral pond into the potential space between the optic nerve and its vaginal sheath—a space which is in direct continuity with the intermeningeal spaces. An excess of fluid thus occurs, of course, in the intravaginal space, and a collection of fluid around the nerveheads.

The dilated vaginal sheaths can be demonstrated postmortem in cases where papilledema has been seen during life. The degree, or indeed often the existence of swelling of the papilla, depends enormously upon the formation of the eyeball. In hypermetropic and emmetropic eyes, the ocular ending of the vaginal space is directed sharply towards, and in myopic eyes away from, the optic nerve—an anatomic plan which in the latter condition, myopia, produces an infiltration of the fluid contained in the sheath of the nerve through the sclera; while in the former, the papilla grows quickly dropsical. The water jacket thus surrounding the optic nerve and collected as a swelling on the papilla, interferes with the venous return through the optic nerve vessels and by doing so produces the tortuosity of vessels and blurring of outline of the disc which we associate with the appearance of papilledema. It is, therefore, of course improper to call these phenomena optic neuritis, which, if a correct term, should be accompanied by great loss of visual acuity. Such loss appears

only when the papillary albuminous fluid has assumed organization and has begun to contract the nerve fibers directly, producing the progressive concentric contraction of the fields characteristic of secondary optic atrophy.

I have, perhaps, wearied your ears with this twice-told tale, but a consideration of these phenomena seemed proper before speaking of the effects produced by massive expanding lesions of the *frontal lobes.* These are often hard of diagnosis, because of our vague knowledge of the function of these areas. The frontal lobes have been generally thought to be the special instrument of intellect, but it is disconcerting to find how much of their cortex can be removed without definite alteration occurring in the memory, attention, or judgment of the victim.

An inapt jocosity may be found; more often drowsiness, morbid sleep and, most importantly, careless incontinence of urine with apparent retention of intellectual function. Slight motor palsy may occur, through pressure backward on the precentral gyri; a moderate degree of aphasia may appear in appropriate persons. This picture, so far, is vague, but I want to direct your attention to a syndrome which I had the chance to describe a dozen years or so ago, which gives exact diagnostic indication of certain expanding lesions of the frontal lobes, and at the same time is easy, especially for the ophthalmologist, to elicit. This is the occurrence of true retrobulbar neuritis with the formation of a central scotoma and primary optic atrophy on the side of the lesion, together with concomitant papilledema in the opposite eye. The macular bundle subserving central vision, physiologically more delicate than its fellows, has presumably a more fragile structure than they, though Brouwer ascribes this fragility to their phylogenetic youth.

Direct pressure on the optic nerve has, as a first result, a deterioration of function, and later a primary atrophy of the macular fibers, with loss of central vision; and this, in spite of the fact that the stouter strands retain functional activity though subjected by their external position to more immediate and direct injury.

This sequence of events is that produced when, from the beginning of disease, one or the other optic nerve is directly pressed upon. If, however, the expanding lesion begins in the middle of the frontal lobe, not involving the underlying optic

nerve, but of course producing a general rise of intracranial pressure, there will be produced bilateral choked discs without scotoma or diminution of vision. The tumor or abscess (in one of my cases the lesion was an aneurysm of the right internal carotid artery,) grows till at last the upper surface of the ipsolateral optic nerve is directly pressed upon. Then, in this eye, edema will quickly disappear and vision deteriorate; a central scotoma will appear, and in a few days' time, temporal pallor will be seen by the ophthalmoscope, an expression of atrophy which later will be present in all four quadrants of the disc. The disc on the opposite side from the lesion will, in the meantime, remain swollen and the vision good. Owing to the proximity of the olfactory bulbs to the optic nerves, it almost always happens that injury to one means damage to the other. Therefore, it is usual in this syndrome that depression or loss of the sense of smell is found on the side on which retrobulbar neuritis has occurred.

Tumors or abscesses of the occipital lobes are, of course, distinguished especially by homonymous hemianopic defect in the opposite visual field. Primarily subcortical growths will cause interference with optic radiations and crossed homonymous hemianopia; primarily cortical lesions of the occipital pole, especially implicating the calcarine fissure, will cause visual Jacksonian fits in the contralateral field, followed usually by deterioration or loss of function in that field; consequences of irritation which, at first temporary and post-epileptic, become permanent when pressure has succeeded in destroying tissue. The subjective phenomena produced by irritation of the calcarine cortex are quite crude and lack constructive quality. For instance, one of my patients, a child with an old hemorrhagic cyst in the left occipital pole, used to cry out "I see the twinkles," by which she meant flashes of light occurring at first in the temporal periphery of the right visual field. These twinkles increased in violence and number, until in some minutes they filled the entire right field. A right sided motor fit then usually took place, with occasional loss of consciousness. On recovery, there always was found a right half blindness for form which, lasting some hours, was succeeded by hemiachromatopsia. Another patient, proven later to have a gumma infringing on the left angular gyrus, had attacks in which he would describe the street traffic on his right as being seen through sheets of

multicolored rain. Temporary alexia would follow such seizures. Visual disorientation occurs only when both occipital lobes have been injured, an improbable result of tumor or abscess growth, but one which was not infrequnetly seen during the war as a result of occipital fracture and bullet or high explosive wounds.

I might confess here, that I have never been able to elicit Wernicke's hemianopic pupillary reaction, nor, indeed, have I met any neurologist in any better case in this matter; for localizing signs in the occipital zone, I have had, therefore, to rely on the phenomena just described, on neighborhood symptoms, and on the type of quadrantic defect produced in the contralateral field.

Tumors of the temporal lobe, and especially of the right temporal lobe, are usually considered the most difficult of all growth to recognize, because of the comparative latency of the regions affected. Just for this reason, however, surgical procedure in them are the less grave; on this account, and because the temporal lobe is so frequently the seat of abscess formation from inflammation of the middle ear, I shall ask you to allow me to speak of the symptomatology of expanding lesions of these areas with some particularity. Because the temporal lobes are vast and uncharted regions with only one known bilateral center, that of taste and smell, placed in the uncinate lobules, and one unilateral center for the storage of auditory memories, in the transverse gyri of Heschl, we are often compelled to look to the pressure effects of temporal lesions on neighboring structures for exact localizing signs. Perhaps my meaning can best be made clear by the consideration of a case in which the diagnosis was very easy.

In November, a woman, aged 51, was admitted to the Neurological Department, Bellevue Hospital, complaining of vomiting, headache, and general convulsions for the previous six months. In August, the convulsion, prefaced by a sudden feeling of fright, was left sided, and was accompanied by an overpowering sensation of smell "like rotten weeds." Later, the smell feeling would come again, she felt funny and different, but with no loss of consciousness. "Then I distinctly saw a woman standing to my left. I was afraid of her. I did not know her. She was dressed in blue; she moved and made motions as though she were talking, but I heard no words." These complex sensations and apparition often came to her,

and a left hemiplegia gradually developed. In October, the left arm and leg grew painful, and to a less degree the left trunk. "If I put my left hand in hot or cold water the pain is terrible." About the same time she noticed "a crawling sort of movement" in the left fingers, which occurred apart from the attacks. The optic discs always were normal. Hemianopia and relative hemanesthesia were found on the left side; in the left arm, there was profound ataxia and athetosis. She was discharged from the hospital, without symptoms, six weeks after the removal by Dr. John Hartwell of a large endothelioma from the posterior part of her right upper and middle temporal gyri.

The convulsive seizures of this patient were typical, beginning always with an aura of fear, referred to the epigastrium and spreading through the central gyri. When the motor fit had exhausted itself, there occurred a true uncinate fit, a crude subjective paroxysmal sensation of a foul odor, with reflex spitting and champing movements of the mouth and lips, with nausea and vomiting. This came often along with that most odd of psychic conditions, a true "dreamy state," in which consciousness, though still held, is curiously altered, and in which the relationship of the patient to the outside world seems changed in a way too subtle for concrete description. In this respect, this woman contented herself by saying she felt "queer," "that everything seemed funny and different," but this failure to portray her defective objective consciousness was almost made up for by her clear narrative of her increased subjective consciousness, namely the complex projection of her submerged memory of a woman dressed in blue, who caused her to be afraid. No doubt these voluminous mental states, as they were called by Hughlings Jackson, often accompany idiopathic epilepsy, but it is certain, that when incoordinated sensory resuscitations of this sort have occurred and organic intracranial disease has been found, the lesion has always been found in a temporal lobe.

I have, at times, speculated on the possible explanation of the interesting fact, that hallucinations of such extraordinary complexity should occur through irritation of this one area. These appearances come to people as a kind of mirage. One of my patients always saw an "old woman dressed in stinking rags and ringing a bell," another saw negroes, and yet another, under my care at present, has visions of people be-

wigged and beruffled in the style of the 18th century crossing her field of vision from the left to the right side. These phantoms are almost always constant for each patient. I have asked biologists if it were possible that the hippocampal lobes, which in lower vertebrates perform some intellectual functions, could at the earliest period of human lifetime carry out primitive psychical functions and act as a storehouse of infantile memory. This hypothesis was based on the fact, that each individual in his development from embryonic to adult life passed through successive stages analogous to those through which the race had passed.

According to this idea, memory pictures might be laid down in a very early period of life in the temporosphenoidal lobes, and in a later period a more highly placed mechanism, perhaps in the frontal areas, might be put to the same purpose.

These early memories would pass into subconsciousness and only occasionally be facilitated to consciousness and in the presence of gross irritation of this area. Such incoordinated resuscitations of memory pictures of a long past and forgotten time of life may account for the phenomenon "deju vu"—that odd sense of having formerly lived through identical experiences which has come often to everyone, references to which are scattered throughout our literature, and which to some are an intimation of immortality in the past as well as in the future.

We must notice again, that when subjective visual spectra occur as a result of temporal disease, they have a constructed coordinated character entirely absent from those crude scintillations which come from irritation of the calcarine cortex. Oppenheim asserted that crude sensations of taste and smell may also result from direct lesions of the olfactory tracts. From this I dissent utterly. Such a contention ignores all known physiologic facts; from such premises we might look for multicolored visual fits from injury to the optic nerves by sphenoidal sinusitis or subfrontal growths, and focal convulsions from internal capsular hemorrhage.

More apt was his observation of fixed pupils in patients suffering from temporal pressure.

This occurs, though rarely, when a growth, placed deep in the lobe, invades secondarily the region of the corpora quadrigemina. In one of my series, limitation of conjugate

upward and downward movements of the eyes was seen during periods when intracranial pressure was greatest, accompanied by transient absences, in the same periods, of the light reflex in the pupils, an Argyl Robertson pupil produced by ingravescent pressure on the quadrigeminal plate.

The optic thalamus is compressed not infrequently in those conditions; it is common to find effects of depression of function of the ipsolateral thalamus in an abolition or marked diminution of emotional expression on the opposite side of the face. It is unusual to find so complete a thalamic picture as was present in the woman just described. The posterior position of the growths probably explained this greater incidence of pressure on the thalamus, as it did the contralateral hemianopic defect. Pressure on the pulvinar or on Meyer's loop of the optic radiations often produces minor quadrantic hemianopias in the opposite visual field.

In the hemiparesis produced by temporal expanding lesions, weakness is most marked in the face, less so in the arm, and least of all in the leg.

The question of the speech defects in these conditions now must be considered. The effect of gradually expanding cerebral lesions on the various centers concerned in our communication with the outer world are widely different from those coming from sudden vascular lesions produced either by experiment or Nature. In these vascular lesions, the damaged area is sharply delimited, and within its borders destruction is usually rapid and decisive; in the expanding cerebral lesions, however, a gradual deterioration of function takes place, producing abnormal speech conditions, less obvious but still explicit.

I emphasize this, because text books still say that word deafness is produced by tumor or abscess in the left temporosphenoidal lobe. This statement is quite misleading, in that such a situation can only be accomplished by destruction of the transverse gyri of Heschl and the posterior three-fifths of the superior temporal convolution; such destruction never occurs except in terminal stages, when exact diagnosis is academic.

Differences in size, situation and rapidity of spread of left sided massive lesions in the temporal lobes will of course produce differences in speech affection, but in them all one feature is constant: a depression of the power to recollect names,

especially those of persons, places and things; this discrimination is accounted for by the fact that our conceptions of persons and things are less closely connected with their names than are the abstractions of their circumstances and properties. Thus, one patient when shown a cigar said, "I know that; I smoke that," when shown money said, "good to have," and an envelope was called by another "something to put a letter in." In these, as in most other cases, there was no loss of word memory; the memories were unimpaired, but in a degree submerged and capable of being brought up to consciousness only by a great effort of will, or by the aid of an additional cognate auditory or visual stimulus. Often such a patient has no speech defect in ordinary conversation, but makes many mistakes when asked to name familiar objects shown to him. This condition is probably the most characteristic of all the temporosphenoidal speech defects, and depends on a degradation of function in the association tracts within the visual and auditory centers. These naming errors are instantly perceived by the patient and annoy him. The right word is recognized as soon as it is heard, and so far are these people from being word deaf, that an incorrect prompting is invariably rejected.

Tumors of the corpora quadrigemina almost always arise from the pineal body; therefore they are commonest in children and adolescents. Such persons have a precocious puberty, the secondary sexual characters may thus appear before the age of ten years; the endocrinologic picture is therefort the reverse of that produced by certain neoplasms of the pituitary gland. These tumors compress but do not infiltrate adjacent brain tissue, and may completely cork the passage between the third and fourth ventricle. The midbrain pressure thus produced gives rise to huge dilatation of the lateral and third ventricles with tremendous papilledema, and to palsies of conjugate deviation of the eyes; usually at first there is interference with upward and down movement of the eyes, but after the involvement of the oculomotor nuclei and the posterior longitudinal bundle, there may be complete external and internal ophthalmoplegia. Of course, the gait is ataxic and there is a gross motor incoordination of the arms, of cerebellar type, and there is evidence of bilateral pyramidal tract disturbance. The enormous dilatation of the ventricles, by ballooning downwards of the floor of the third ventricle on

the optic nerves, may cause sudden blindness early in the history of the disease.

These patients are usually very sleepy, and in view of our experience with epidemic encephalitis, where the lesion is periaqueductal in position, we may look on such morbid sleep as a focal symptom of subthalamic disease, quite apart from the pressure conditions produced by such disease.

Massive lesions of the pons are usually not hard to distinguish, owing to the large number of focal points represented therein. Combinations of ipsolateral palsies of the fifth, sixth and seventh nerves occur, together with motor paralysis of the opposite limbs. It is important to determine the nuclear origin of these palsies, in that abducent lesions so often come from rise of general intracranial pressure. In such cases, however, they will not be accompanied by ipsolateral facial paralysis, nor will the movement of the contralateral internal rectus be reduced in amplitude or strength. Pontine gliosis may be diffuse and give the appearance of simple pontine hypertrophy; in such instances, damage is apt to be two sided and papilledema very late in appearance. The sensory fillet is amazingly resistant to compression, and objective sensory change from pontine lesions is consequently rare. Lesions of the crus are often conglomerate tubercle, and are characterized by an ipsolateral third nerve paralysis, with ptosis of the eyelid and characteristic defects of extraocular movements. The red nucleus and the rubrospinal tract damage produces a coarse tremor in the contralateral hand.

Tumors of the cerebellum are characterized by dizziness and unsteadiness of gait; by early choked disc; coarse, slow nystagmus on looking to the side of the lesion, and fine more rapid nystagmus on looking away from the lesion; ipsolateral ataxia of arm and leg, and a tendency to fall towards the side of the tumor. A curiously tilted attitude of the head seen often in these cases is probably of vestibular rather than of purely cerebellar origin.

Tumors growing in the cerebellopontine angle most often arise from the endoneurium of the acoustic nerve; the first manifestation is tinnitus and vertigo, followed by progressive nerve deafness. The trigeminus is next involved; subjective numbness and tingling in the face and side of the tongue. Palsy of the motor root is characteristic of a tumor of the base rather than that of the cerebellar angle. By the time the

seventh nerve is involved, definite evidence of cerebellar compression is forthcoming. These growths grow more slowly than those of the cerebellum itself; often two to five years may pass before successive implication of contiguous structures reveal the diagnosis.

The hypophysis is often the seat of tumor growth, to be recognized by neighborhood signs and those of altered pituitary function. The former include most typically bitemporal hemianopia, but almost all forms of hemianopic defect may occur, especially in the earlier stages of the disease. Papilledema is rare and occurs only when the mass has blocked drainage by invading the third ventricle. Primary optic atrophy is the rule. Headache is severe through distentions of the gland capsule. Adiposity, eunuchism and abnormal sugar tolerance may come from defective secretion; acromegalic signs are rarer and result from a simple adenomatous hypertrophy of the anterior part of the gland.

In the early discovery and appreciation of the meaning of quite small defects of function, correlated with the known facts of intracranial anatomy and physiology, lie the secret of success in this diagnostic field. These defects of function in their beginning often come under your view rather than mine, and I appreciate greatly your kindness in allowing me to talk to you on such matters and your generosity and patience under so lengthy a sermon; he who cares for patients suffering from brain tumor must bring to his task thought and stout action;—and there is need of a formidable optimism, for the dice of the gods are loaded.

DISCUSSION.

DR. WILLIAM L. BENEDICT, Rochester, Minn.: In the diagnosis of intracranial expanding lesions including neoplasms, aneurysms, and cysts, the functional testing of the eyes plays a very important part. Probably more important is the aid given to localization of the intracranial lesions as furnished by visual fields, ophthalmoscopic examination of the fundus, and disturbance of ocular motility. Fairly definite syndromes have been established for the localization of lesions situated in the region of the chiasm, midbrain, and temporal lobes. With less certainty, lesions of the frontal and occiptal lobes often can be located by the aid of ophthalmologic examination. To take them in the order in which Doctor Kennedy has discussed them, I shall refer to the ocular phenomena, leaving the discussion of the other symptoms and effects of the lesions to my colleague, Doctor Loeb.

In a series of 168 cases of known cerebral tumors examined at the Mayo Clinic from 1919 through 1924, 60 were located in the temporal lobes. Of these, 57 were operated, and the localization of the other three was correctly diagnosed as proved by autopsy. In the series were 53 infiltrating gliomas, 3 dural endotheliomas, 2 calcified hematomas and 2 angiomas. In contrast to lesions in the neighborhood of the chiasm, lesions in the temporal lobes do not produce changes in visual acuity early. In only 2 of our cases, in whom the fundus was practically normal in appearance, was there moderate reduction in vision, and one of these cases showed a mild pallor of both discs. In 12 cases, there was a decrease in visual acuity due to secondary changes in the nerve from long continued papilledema or consequent atrophy. 32 of these 60 cases had normal vision. In 9 cases the vision could not be tested by ordinary means. Again, in sharp contrast to the lesions in the neighborhood of the chiasm is the prevalence of choked disc in tumors of the temporal lobe. In 45 cases, or 75%, papilledema ranging from ½ diopter to 7 diopters was recorded. This papilledema was equal in the two eyes in 24 cases. One case had a definite bilateral secondary optic atrophy without edema when first examined, thirteen cases had normal appearing discs, and one case had a mild pallor of both discs. There was no relation between the eye showing the greater papilledema and the side in which the tumor was located. The position of the tumors was nearly equally divided—34 situated in the left lobe and 26 in the right lobe. Papilledema evidently is an early and quite constant finding in temporal lobe lesions in contrast to chiasmal. Quite characteristic changes of the visual fields have been noted by Cushing and by Lillie.

In 1921, Cushing reported 59 verified temporal lobe tumors, of which he was able to chart the visual fields of 39. Of these 39 individuals, homonymous field defects indicating involvement of the temporal loop of the optic radiation were present in 33 instances. Eight of these cases showed homonymous hemianopsia, while 25 showed partial hemianopsia, with defects more or less quadrantic in character. In our 60 cases, changes in the visual field were found in 52. In 8 cases, the fields were normal both for form and colors. In 3 of the eight cases having normal peripheral fields, the blind spots were enlarged, due to papilledema. In the other 5, the discs were normal. Seven of the cases were infiltrating

gliomas, and one was a calcified hematoma. There were 10 cases showing quadrantic defects for one or more colors, the form field being normal. The color defect occured in the upper quadrant in 6, and in the lower quadrant in 4 cases. Homonymous quadrantic defects for form and color were present in 15 cases, 9 in the lower quadrant, 6 in the upper quadrant. Homonymous hemianopsia for colors with form fields normal was present in 3 cases, while complete homonymous hemianopsia for form and colors was found in 15 cases. In this series, 17 cases were definitely localized by the perimetric fields, while the neurologic findings were only general in character. In contrast, 15 cases were definitely localizable by the neurologic findings while the ophthalmologic examination was of no particular localizing value. Six of these had normal visual fields, while in 9 cases the visual fields could not be obtained, due either to the mental condition of the patient or to his lack of vision. Definite localization as to the site of the lesion was possible in 43 of the 60 cases, due to definite field changes alone, while definite localization from the neurologic examinations alone was possible in 40 of the 60 cases. Ocular motor changes consisted in paresis of the left abducens in 6 cases, ptosis of the left upper lid in 2, partial external and internal ophthalmoplegia 2, and convergence weakness in 3 cases.

Lillie has called attention to the prevalence of ocular motor changes in the left side even though the tumors were about equally divided in the right and left lobes. Doctor Kennedy has called attention to the visual hallucinations in patients showing temporal lobe tumors. Cushing recorded visual hallucinations as found in 13 out of 59 cases. In the 60 cases examined at the Mayo Clinic, not one had visual hallucinations of any type. Combining the two series of cases, visual hallucinations occur in less than 10% of proven temporal lobe lesions. The most characteristic ocular changes found in temporal lobe tumors occur in perimetric fields. Homonymous quadrantic defects for either form or colors or for both occured in 43 of 51 cases in the Mayo Clinic series. In Cushing's series, 33 of 39 cases showed homonymous field defects. As Lillie has pointed out, if we add to Doctor Kennedy's syndrome of voluminous mental states, visual hallucinations, uncinate fits, speech defects and choked discs, the characteristic changes of the peripheral fields, namely quadrantic homonymous defects for form and color, a rather definite and complete syndrome for temporal lobe tumors is established.

No such characteristic findings in either the visual fields or the fundus occur with regularity in tumors of the frontal lobes. Papilledema is usually a very late finding in frontal lobe tumors, except in cases where the tumor is situated near the base or in close proximity to an optic nerve. Papilledema results not only from increased intracranial pressure, but from direct pressure upon the optic nerve anterior to the chiasm, either within the cranial fossa or within the orbit. There is no doubt that paraneural tumors anterior to the optic foramen will produce typical choked disc in one eye. Tumors of the frontal lobe, when situated near the base on either side, may produce a unilateral choked disc. In later stages of the growth of the tumor, this choked disc may be followed by optic atrophy before the development of choked disc in the other eye from increased intracranial pressure. Doctor Kennedy states that a common finding in tumors of the frontal lobes is an ipsolateral optic

atrophy with contralateral choked disc. He also states that the atrophy is due to typical retrobulbar neuritis. It is well known that optic atrophy follows choked disc, and that after an optic nerve has once become atrophic, the disc does not become noticeably swollen. Optic atrophy also occurs as a result of direct pressure by a growing neoplasm on the optic nerve. It is quite probable that the ipsolateral optic atrophy of which Doctor Kennedy speaks is a result of direct pressure exerted by a neoplasm, and the atrophy may or may not have been preceded by papilledema. I think it is not necessary to regard the atrophy as subsequent to an optic neuritis. We should more clearly differentiate optic neuritis and inflammatory conditions of the nerve from choked disc or papilledema, which is a stasis phenomenon.

The visual field changes found in cases of tumor of the frontal lobe are not characteristic. They depend upon the amount of damage done to the optic nerve by direct pressure or through choked disc. In 2 of our cases of frontal lobe tumors, the chiasm was affected, giving a bitemporal hemianopsia with changes characteristic of a lesion at the chiasm; as these cases also had choked disc, the chiasmal syndrome was complete.

Tumors situated in or near the chiasm produce ocular phenomena that have been so thoroughly studied and so widely discussed, that their diagnosis presents less difficulty than most other intracranial lesions from an ophthalmologic standpoint. In our series of tumors involving the optic tracts near the chiasm, 60 of 64 patients had noticeable changes of visual acuity of one or both eyes. In contrast to tumors of the temporosphenoidal areas, reduced visual acuity is a common early symptom of chiasmal lesions. In the early stages of the growth of the tumor, notching of the superior temporal fields occurs quite regularly, but often homonymous hemianopsia or homonymous quadrantic defects of the fields may be found. Macular vision is disturbed in a very large percentage of cases. In keeping with this difference in macular involvement, scotomatous field changes were at one time or another continuous with the physiologic blind spot in practically all cases, and the blind spot was almost always incorporated with the temporal field defects either for form or for colors. The ophthalmoscopic examination of the fundus also is important in differentiating chiasmal and temporal lobe lesions. Whereas choked disc is a common finding in tumors of the temporal lobe, it is a rare finding in tumors situated near the chiasm. The characteristic change in the discs in chiasmal lesions is a waxy pallor of the disc without loss of substance, which precedes optic atrophy by many months or many years. This finding, to which I called attention in a paper on the "Early Diagnosis of Pituitary Tumor with Ocular Phenomenon", read before the section of Ophthalmology of the American Medical Association in 1920, has been verified by our own additional findings as well as by observations by other examiners. Chiasmal lesions not of pituitary origin give changes in the visual fields and optic discs which often cannot be differentiated from changes produced by tumors of pituitary origin. As a group, they can be differentiated from tumors of other locations if characteristic ocular changes can be elicited.

Dr. John Gordon Wilson, Chicago: One remark in Dr. Kennedy's paper I think ought to be emphasized, that is, that if we want to be accurate in our early diagnosis of intracranial lesions, we must base our

findings on the anatomy and physiology. It must be obvious to everyone that the mixup we have in regard to intracranial lesions and their symptoms is getting us nowhere.

He made another remark which to me is very interesting, that is, that probably the temporal lobe is the most primitive part of the being in ontogenous development. Remember that the ear is the most primitive part of the body, and the embryologists tell us that the first part they see of the human embryo, that which gives them position of the embryo, is the human ear, then the eye. That raises this thought—is the auditory function in the temporal lobe? I have an idea also that somewhere about the temporal lobe there is some function connected with balancing, and if so this idea of Dr. Kennedy's may be pushed too far, although it is interesting.

Information derived from otologic tests are in themselves seldom sufficient to localize brain lesions. With our present knowledge, even without available aids, a diagnosis of intracranial lesion should be always correlated with details from other parts. With all our otologic tests of deficiency of reaction, we rely chiefly on the alteration of normal eye movements. This is not to be wondered at, when one thinks of the ease with which the eye balances. The labyrinth reflex to the eye is more direct, and compared with pointing, is influenced less by mental operation, and so less liable to lead to false interpretation. The rotation tests are not of equal value to the caloric. When we have a means of picturing nystagmus during rotation, we shall find them superior, but so far as rotation goes, I find more valuable stimulation with cold water. Valuable experiments are now being carried on, in which attempts are being made to photograph all the movements during rotation, and if these are successful, they will be an immense advantage to us in the diagnosis of intracranial lesions.

One other general point is this—in rotation tests, the reaction is much more pronounced in cerebellar than in cerebral lesions. This is especially well marked in the eye. If you have a lesion in the region of the lateral ventricle, you almost always get an excessive reaction from rotation tests; whereas you get less in cerebellar lesion. And you certainly get more vomiting and discomfort.

The two ear functions are the vestibular and the acoustic, and any intracranial lesion thus may be effected at the periphery, and pressure at the periphery includes something that is comparable to what takes place in the eye in choked disc. In the early stages both these symptoms are shown, as one would expect them, by changes at the threshold of stimulation. Now we have in the perimeter test a means of charting the visual field, and thus we gradually come to some knowledge of the early threshold changes from early choked disc or pressure along that particular point. It has not been so easy to find the threshold of stimulation of the vestibule, but it has been done to some extent. The little knowledge we have of it makes us know that there come in errors that lead to false interpretations. I will give you one example. One of the great puzzles during the war was the fact that when the aviator turned upside down, we found this change. The explanation is that at a certain rotation, the stimulation of the labyrinth is at the threshold and at that particular space errors constantly occur. In this respect the work that has been done that is really important is by Dr. Dodge, psychologist at Yale.

Coming now to the question of nystagmus—this test concerns primarily the adjustment of the head to the body and to the outside world which we call, properly, balancing. The most delicate balancing mechanism of the head, which is to be kept adjusted to the outside world, is the eye, therefore it is often very early affected. A great part of the balancing of the eye comes from the ear, and the next most important part comes from the eye muscles. Disturbance in any pathway may give you a disturbance of the eye muscles, and we call that oscillation nystagmus. There are two varieties, ocular and labyrinthine. The ocular is a pendulum movement, whereas the labyrinthine is quick and slow. Vestibular nystagmus, pure and simple, is always slow to the side of the defect and rapid to the other side—a slow to the labyrinth, and quick to the cerebral. There is one modification of the vestibular which has received very little attention, and that is the cerebellar nystagmus. If a patient turns his eye to the side, you get a tremor and you can always be sure it is a cerebellar ataxia. The vertical nystagmus never occurs in connection with a labyrinth lesion.

One reason why the caloric reaction is much preferred, is that in the reaction that takes place in the labyrinth, the eye is very important and we know the pathway. We know it goes along the vestibule and through the pons up to the eye muscles. That is a slow movement, and the study of that should give us much more information than we are now obtaining. The quick movement is something added, something that only comes when it is preceded by the slow movement. You ask, "Does the quick movement ever drop out?" Yes. "In what lesions?" In lesions which are above or near the midbrain. Let me give you an example. The quick movement dropping out is much better seen in man. We had a man who came in for operation on the left temporal lobe. After a period of days, cold water was put in the left ear. The result was a deviation of the eye and no quick return. Cold water was put in the right ear, and the result was a very typical nystagmus. That has been seen repeatedly in man. Very often these cases have been near the temporal lobe. And we often see such a case as this—an injury to the brain; cold water is put in the right ear and we get a distinct nystagmus, slow to the right. Hot water is then put in, and instead of getting a reversal, we get slow to the right. I do not understand how it takes place, nor just where it takes place.

One word in regard to the question of separate pathways. You know it has been argued, that when the pathway from the vertical and the pathway from the horizontal canals get to the junction of the pons and the medulla, they separate, the vertical going through the cerebellum, and the horizontal straight on to the pons. That is based on the following—in any case of pressure tumor in the posterior fossa, you get no reaction if you put cold water in a patient's ear; but if you put his head back 90 degrees and stimulate the horizontal canal you get a reaction. On that basis, you have a differentiation of parts. As we have no knowledge, anatomic or experimental, that such a differentiation or separation of parts occurs, it is unnecessary to use that explanation of what is seen clinically, because in a great majority of these cases, if, after you have stimulated the horizontal by putting the head back, you tilt the head up, you get a reaction from the vertical. In other words, what you have is not an obliteration of the pathway, but a delay in the functional reaction along

the pathway. Also, it is known from experimental work, especially that done by Maxwell and Cole, that the horizontal is much more easily stimulated than the others, and psychologically and physiologically we know that once a pathway is opened up, it is much easier to get stimulation to travel along that particular pathway.

DR. ARTHUR W. PROETZ, St. Louis: I was especially interested in Dr. Kennedy's remarks about the effects of intracranial lesions on the olfactory tract. I have been occupied for some time in making olfactory measurments, qualitative and quantitative, in an effort to try to localize certain brain lesions. Of course, I have not been successful so far in doing that, but I have made some very interesting observations. Among them was this—that in certain intracranial pressures we got complete anosmia to certain odors alone, and not to other odors. So far, we cannot say what that means, because these cases are rare and we have not been able to check them up, but I would be glad to know if Dr. Kennedy or anyone else has made any such observation. We have a case now who is completely anosmic to phenol and benzol. The odors of these two stimulants are nothing alike, still there is a close chemical relation between them. Where the tumor is I do not know, because the pattient has not been operated upon, but I would like to know if anyone else has any such data.

DR. EDMOND E. BLAAUW, Buffalo, N. Y.: It has struck me from the ophthalmologic side, that the question of nystagmus has not been touched. There is a form of nystagmus that is called nystagmus refractorius, that points at a definite syndrome of pressure at the entrance of the aqueductus Sylvii. I do not know whether the Doctor has noticed that. Elschnig has published something about it.

This paper is interesting because it brings the neurologist, and the ophthalmologist and the otologist together.

The Doctor said he never had had a hemianopic light reaction of the iris. I wonder if he ever used the instrument of v. Hess—the hemikinesimeter.

DR. FOSTER KENNEDY, New York City, (closing): I am very sorry that I did not know of the existence of the instrument Dr. Blaauw mentions for getting a pencil of light into the pupil. I probably will not be able to afford it, but when I get back to New York, I will see what I can do about it. If I succeed in getting one, I shall be the first neurologist on record to have one, and I am very much indebted to you.

Dr. Benedict spoke of the enormous percentage of temporal lobe tumors which he found in his series, and I am sorry for him that he did not find more visual projections in his cases, because they are interesting to watch and exceedingly dramatic. I can only conclude that if there be anything in my theory, Dr. Benedict's patients must have led a very vacant, infantile life!

I think Dr. Benedict and myself are at one on the question of the kind of optic atrophy which occurs in subfrontal lobe tumors. My notion is, that when the tumor begins on the under part of the frontal lobe, it compresses the optic nerve from its inception; then the optic nerve suffers a retrobulbar neuritis, and we have the syndrome of socalled primary optic atrophy. If, however, the tumor is not immediately on the subfrontal lobe, but is in the middle of the lobe (in cases of bilateral papilledema), when the tumor reaches the sublying optic nerve, the

papilledema on that side is obliterated and retrobulbar neuritis and rapid blindness takes its place.

I am chagrined that Dr. Wilson thought the ear had not received enough attention, but I may explain by saying that I did not get as far south in my examination of the brain in the course of the paper as the cerebellum and ventricular apparatus. There will be more in my published remarks regarding this but not so much as Dr. Wilson has told, because I do not know as much. I have, however, had considerable experience with acoustic tumors, which are usually benign, and in which the character of the nystagmus is of first rate diagnostic importance. I quite agree with Dr. Wilson that vertical nystagmus is never labyrinthine; it is very frequently seen in epidemic encephalitis where the lesion is midbrain, and one of the most important diagnostic features of periaqueductal inflammation is very rapid "electric like" vertical nystagmus.

I was interested in Dr. Wilson's statement that the auditory apparatus was one of our oldest structures. I think that is quite true as regards the vestibular apparatus, but I doubt if it be true of our auditory apparatus.

Dr. Wilson: What I said was this—that in human embryo, the first spot the embryologist can put his finger on is the ear—the outside of the ear—and the mechanism of the brain corresponding to it.

Dr. Kennedy: Of course, when we were fish, we had a double line of differentiated epithelial cells which reacted to rhythm, and which told us whether we were swimming upside down or right side up. I suppose really the antiquity of the vestibular apparatus and its proximity to the cochlea has something to do with the affection for the "jazz" rhythm in music on the part of primitive peoples!

I am very sorry to say that I have no knowledge whatever on discriminating anosmia. I have always used rule of thumb methods in testing the olfactory sense. I shall try to modify and improve my technic in the future.

A written question has been sent up—asking if I have observed loss of word memory in lesions of the right temporosphenoidal lobe in left handed persons. Yes, I have. If a left handed person comes from left handed stock, i. e., if he is not a "sport," then his mechanism of speech will be on the right side of the brain. If a left handed person, however, be a "sport" in his stock, if there be no other left handed person among his collaterals, it is not wise to be sure that his speech center is reversed from normal. His speech center will usually follow that of his stock rather than that of his own individual handedness.

DIFFERENTIAL DIAGNOSIS OF LATERAL SINUS THROMBOSIS.*

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The object of this paper is to review briefly the symptomatology of lateral sinus thrombosis, and to present for your consideration the results of our studies of the dynamics of the spinal fluid and its practical application in sinus thrombosis.

Cases of lateral sinus infection have apparently been on the increase during the last few years, due either to our increase in knowledge resulting in more frequent correct diagnoses, or to the type of infection which has been prevalent since the "flu" epidemic of 1918.

It is presupposed that there has been, within a relatively short time, or that there is present an infection of the middle ear itself, or middle ear and mastoid. We have never observed, nor are we able to find authentic instances of lateral sinus thrombosis in the absence of aural infection, excluding fractures of the skull and meningitis. Trendelenberg, in 1908, first brought to our attention a case of septic thrombosis of the jugular vein as a result of pharyngeal infection. This was again brought to our attention by Dr. Mosher in 1921.

The usual, and fortunately the majority of cases of acute mastoiditis, under proper care and operative procedures, run an uneventful course through their convalescence without evidence of septic complications, as shown by observation of the clinical charts in any of our large aural institutions. This fact is so striking, that after the very frequent reactionary temperature during the first twenty-four hours following operation, any subsequent rise in temperature is immediate cause for anxiety and thorough investigation.

Should evidence of complications occur, what are the problems to be considered? First, investigation of local conditions.

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An accidental folding of the auricle will not infrequently cause a rise in temperature of several degrees. This is especially true in small children. It is accompanied by distinct local discomfort. A slight rise in temperature accompanied by very severe headache is often accounted for by a very tight compressing bandage. The local reaction, and this is especially true in the acute fulminating type of mastoiditis, is accompanied by redness, extreme tenderness and edema of the tissues adjacent to the mastoid incision. This is usually readily relieved by warm moist packs. The presence of, or the previously existing furunculosis of the canal is occasionally observed. This is associated with pain and localized swelling and tenderness. Where iodoform gauze has been used, it is not infrequent to have a temperature of several degrees, often associated with chills and local discomfort. Examination of the wound reveals a very marked dermatitis, often indurating in type. Erysipelas is often associated with temperature and chill occurring twenty-four to forty-eight hours before any demonstrable local manifestations are present. Its evident development, as a rule, involves the auricle, the pre- and postauricular regions, very frequently extending up over the cranium, and shows a definite outline which does not occur in iodoform poisoning. The presence of acute cervical adenitis, which is not at all infrequent in children, must also be seriously considered as a causative factor. Thorough physical examination of the patient must be made, bearing in mind the possibility of a pneumonia, and, in children, not infrequently the presence of a central pneumonia without demonstrable physical signs; acute endocarditis with or without demonstrable physical signs; any acute abdominal complication; the presence or absence of an acute nephritis. The most common causative factor of postoperative temperature in children in the presence of pyelitis. This latter is surprisingly frequent, not only in our public institutions but also in private practice, since infants and small children are very prone to develop a pyelitis in the presence of any septic focus. Postoperative cases are peculiarly prone to develop an acute pharyngeal or tonsillar infection, and it is not infrequent that a wave of this infection will apparently develop in a ward within 24 to 48 hours. The probability of the development of an acute otitis media of the other ear is often overlooked.

On the occurrence of unusual symptoms during the course of aural infection, a thorough and careful observation and examination having eliminated the above mentioned factors, the intracranial complications must be considered,—brain abscess, meningitis, infection of the sinuses. We will not consider brain abscess in this connection other than to mention the fact, that an acute cerebellar abscess as a rule presupposes lateral sinus infection. The development of distinct suppurative meningeal symptoms is as a rule characteristic. It must be borne in mind, however, that many cases of sinus infection demonstrate clearly symptoms of serous meningitis during the very early stages, symptoms such as are usually designated as meningismus. It must also be remembered that in a large number of our fatal cases, the suppurative meningitis is secondary to or accompanied by infection of the cranial sinuses, especially of the superior and inferior petrosals, and we are unprepared to make a statement as to whether many of these cases are primarily sinus thrombosis or are secondary to the meningeal infection.

Contrary to the preconceived idea of many authors and of many otologists, I wish to impress upon you, that it is impossible to designate any definite cardinal symptoms which may be considered didactically as distinctive of lateral sinus infection or thrombosis. We must consider the symptoms present in an individual case as a symptom complex. Occasionally, we encounter a typical text-book case, but as you know, the symptomatology and pathologic findings vary not only as to the personal equation of the individual, but also as to the prevailing cycle or epidemic occurring at the time.

We have observed definite cycles of infection occurring at intervals and demonstrating clinical and physical phenomena which are didactically different. We must be prepared to recognize these cycles and to deal with the prevailing type of infection and its sequelae. In the acute septic explosive type of sinus infection, we have a very sudden rise of temperature to the maximum, often to 105-106 degrees, with an equally rapid recession to normal or subnormal, occurring once, oftentimes 3 or 4 times during 24 hours, associated with distinct chill and sweating, a very evident picture of acute sepsis. This may continue indefinitely, or it may occur in periods of intervals of 2 to 3 days. This type is usually associated with distinct symptoms of meningeal irritation, as headache, slight

stiffness of the neck, and evident involvement of the superficial reflexes. We have often observed a positive Kernig sign. The positive blood culture may or may not be present, since apparently bacteremia varies decidedly in the individual case, being present on one culture and absent on the next; when present it may be considered to a certain degree confirmatory, when absent negative. The leucocytosis is usually increased to a more marked degree than in any other of the types.

The case most frequently overlooked, or not recognized during the early stages, is the one which shows a persistent irregularity of temperature, at no time rising more than 1 to 2 degrees, and showing no evidence of active sepsis. A patient complains of slight dull headache, inability to sleep at night, sensation of indefinable fullness and heaviness localized to the infected side, a marked lassitude, loss of appetite, and general malaise. Definite rigors are unusual. Close questioning will at times elicit a description of chilly sensations with slight flashes. Blood culture may or may not be positive—more often positive than in other types encountered. Leucocytosis seldom above 15,000.

Again, we have the case which shows an elevation of 2 to 3 degrees of temperature which does not remit to normal, but shows slight remissions and elevations from 100 degrees as a basis. In this type of case the temperature may, at intervals of several days, remit and remain normal for 24 hours to 10 days or more, and then recur. As in the previous type, the patient complains of unusual lassitude, inability to concentrate, dull indefinite sensations of heaviness localized to the infected side of the head. The leucocyte count as a rule varies from 8,000 to 12,000. Blood culture may or may not be positive. The blood culture, when positive, is a fairly definite indication that there is a septic focus, and, other foci being excluded, must be considered as a definite indication for operative interference.

Again, we have the afebrile type. In 1919, we reported 11 cases of this type occurring in a series of 73 consecutive cases. Numerous cases have since been reported by others. From our observation, these all occurred in cases of long standing, and were discovered only during the operation on the mastoid, and, as a rule, associated with a perisinus abscess. This condition occurs much more frequently than we have thought, and as I will show later, many of the cases of perisinus ab-

scess demonstrate more or less obliteration of the lateral sinus. One of the most constant objective symptoms which we have found has been edema and infiltration, with tenderness on deep pressure over the area drained by the emissary vein. This is quite distinct and readily differentiated from mastoid edema and tenderness, and, per se, never extends over the posterior border of the mastoid. This same symptom or sign is occasionally found just below and posterior to the mastoid tip, due to blocking of the posterior condylar vein. This latter is not constant. This edema and tenderness must be differentiated from acute or subacute adenitis. Tenderness along the jugular vein we have never been able to demonstrate to be, when present, due to other than infection of the cervical glands. The refusal of healthy granulation tissue to develop may lead one to suspect an involvement of the sinus.

Optic neuritis, or choked disc, we have found to be present in about 10% of the cases. We have never been able to demonstrate that pressure over the patent sinus, or both simultaneously, has made an appreciable difference in the eyeground. Anatomically, the type of mastoid apparently plays no important role.

After reviewing the well known symptoms with which you are cognizant, I am afraid that we will feel much as Dr. James Babbitt felt, when in a recent address, having carefully summed up the symptomatology, he inserted the following paragraph:

A. "Unfortunately none of the cases in the writer's recent experience has conformed in any material degree to this outline."

B. "Patient seems more ill than could actually be accounted for by associated or previous mastoid symptoms."

Dr. Babbitt has certainly uttered a truism, for up to within recently, we have had no definite test other than actual investigation of the sinus lumen itself.

As the result of several months' investigation, in conjunction with Dr. James B. Ayer, of the dynamics of the cerebrospinal fluid, we feel justified in presenting for your consideration a relatively new test in the diagnosis of lateral sinus thrombosis. This test is based upon the fact that increased intracranial pressure is normally propagated throughout the cerebrospinal fluid system and may be measured by a manom-

eter employed in connection with lumbar puncture. Compression of the internal jugular veins causes increased intracranial pressure with immediate rise in spinal fluid pressure.

It is now generally considered that absence of rise in fluid pressure on jugular compression (positive Queckenstedt[1]) indicates spinal subarachnoid block, a most important and early sign in spinal cord tumor. More recently, it has been shown that block may also occur and be detected in a similar manner in the presence of tumor of the cerebellar fossa.[2]

In both of these conditions, the evidence on which diagnosis of block rests is absence of rise in pressure below the level of obstruction, the increased intracranial pressure being assumed in single puncture below, or proved by two punctures, one above and one below the level of obstruction.[3]

It may be safely said that the criteria on which these tests depend and the results of the tests have been for the most part dependable. So much so, in fact, that the effect of jugular compression on spinal fluid pressure now forms one of the routine tests in diagnostic lumbar puncture.

While the criterion on which a diagnosis of sinus thrombosis is based is the same as that of subarachnoid block, namely, absence of rise in pressure in the spinal fluid manometer, it will be seen that the mechanism by which this is shown is quite different. Instead of an increased head of fluid which cannot be transmitted because of obstruction, there is here no elevation of pressure at its source, because of venous obliteration.

Also, we are here concerned with careful comparison of the effect of pressure on each vein separately, not merely with the effect of simultaneous compression of both internal jugular veins, as in the diagnosis of cord tumor.

Sinus Thrombosis Test.

The test for sinus thrombosis, as we have used it, is as follows: With the patient in the lateral position, lumbar puncture is performed, and the fluid is allowed to run into a glass manometer of 2 mm. bore. The initial pressure reading is noted; also the presence of pulse and respiratory oscillations as evidence of patency of the manometric system. Now, without in any way disturbing the patient, an assistant gently presses on one side of the neck between the larynx and the sternocleidomastoid muscle until he feels a strong carotid

arterial pulsation. During the compression, the operator watches the rapidity of rise of the fluid column in the manometer, the promptness of its beginning and the full height to which it attains, and, on release of jugular compression, the rapidity of drop in pressure. The procedure is now repeated on the opposite side of the neck, and then, for comparison, both sides of the neck are pressed simultaneously.

In a typical case of lateral sinus thrombosis, there is a prompt and rapid rise in fluid pressure to twice or three times the initial reading when the internal jugular vein draining the normal sinus is compressed. This pressure rise is maximal, being equivalent to the pressure attained when both jugular veins are compressed.

Pressure over the vein draining the thrombosed lateral sinus causes either no rise or more commonly, a slow rise of only from 10 to 20 mm. in the manometer.

These findings are characteristic of complete obliteration of the sinus. Partial obstruction from mural thrombosis naturally gives less striking results, yet is of value when correlated with the clinical findings.

We may consider some of the pitfalls of the technic as encountered in the study of 84 cases:

1. Incomplete compression of the jugular vein as the result of faulty technic in applying compression accurately, as a result of obesity, tumors, cervical adenitis, or phlegmon.

2. Absence of rise in fluid pressure or very slight rise is occasionally noted in patients with no obstruction either in venous or in subarachnoid fluid systems when the cerebrospinal fluid is under low tension. This can easily be proven in a given case by injecting from 10 to 20 c.c. of physiologic sodium chlorid solution, whereupon dynamic tests will appear normal.

3. An abnormal rise in fluid pressure may result from carelessness in performing the test, leading to extraneous acts on the part of the patient. If the patient coughs, holds his breath, grunts, or even moves the head or body, a prompt rise in pressure will be noted under normal or abnormal conditions, thus vitiating any record.

4. Normal difference on the two sides. The foregoing pitfalls concern bilateral as well as unilateral venous compression. A group of unselected patients in the neurologic clinic were examined with a view to determining the uniformity in

response to jugular compression on the two sides. The following conclusions are justified from this study: (a) There is often a marked difference in the height of the pressure reading following compression of each internal jugular vein separately. However, a difference of over 50 mm. is unusual and over 100 mm. exceptional. (b) Given a normal initial spinal fluid pressure (150 mm.) the rise on jugular compression of one side usually will be ample, seldom less than 50 mm., more likely over 100 mm. (c) The rise in fluid pressure appears to be somewhat less when the vein on the dependent side is compressed. It is likely that this is an error in technic due to difficulty in reaching this vein rather than an inherent difference in physiology.

5. Anatomic variation in the size of the lateral sinuses must be admitted, although a demonstrable difference in the size of the two lateral sinuses is unusual.[4] This difference is occasionally observed, and in our experience with this test one example of such discrepancy occurred. We must, therefore, admit a certain irregularity in the anatomic disposition of the tributaries of the internal jugular vein, variations of the facial vein being of greatest significance in this test. It is probable that the slight rise frequently observed on compression of the jugular vein on the side of complete sinus thrombosis is to be explained by coincident compression causing stasis in the ophthalmic venous system.

Dangers.

The possibility of two known dangers must be admitted in the use of this test. We refer to (1) sudden death from the pressure of subtentorial abscess or tumor following lumbar puncture, and (2) the precipitation of acute meningitis.

The first danger may almost be eliminated by a careful preliminary examination of the patient. If the neurologic examination suggests the presence of abscess or tumor, but especially if choked discs are found, the well known danger of medullary paralysis following lumbar puncture must be entertained. In such patients the spinal fluid tests should not be attempted.

The second possible danger relates to the liberation of organisms from an area of localized infection or from the blood stream. The former appears to us to be unlikely unless large amounts of fluids are withdrawn. But the latter danger

rests on experimental evidence in which it was shown that in the presence of blood infection, passage of organisms from vessels to meninges may be facilitated by withdrawal of cerebrospinal fluid, by cerebral venous congestion.[5] Although recognizing this as a biologic principle, we have so far failed to note this serious coincidence clinically. Further, we believe that this danger is not great, because we seldom have present in man the setting required for the experimental production of meningitis, namely, septicemia with a sufficient number of organisms or of proper virulence; nor do we reduce fluid pressure so greatly or congest cerebral vessels so long as in the experiments in which meningitis was precipitated from septicemia.

We feel justified, after careful analysis of these cases, in stating that a definite dependable preoperative diagnosis of complete lateral sinus thrombosis, positive or negative, may be expected, and that confirmatory data of mural thrombosis is often demonstrable.

We have outlined the following classifications:

1. Complete obstruction—obliteration of the lumen of the lateral sinus or the jugular bulb, or of both.
2. Incomplete obstruction—mural thrombosis.
3. Negative—no obstruction to venous outflow.

Conclusions.

When, during the course of an aural infection, there is evidence of sepsis, infection of the lateral sinus or jugular bulb must be considered. Evident sepsis being present, a careful differential diagnosis having excluded all other sources, ligation of the jugular and exploration of the lateral sinus is indicated.

When obstruction of lateral sinus, jugular bulb, or internal jugular vein occurs, block may be demonstrated by dynamic studies of the cerebrospinal fluid.

The manometric studies on the fluid have proved reliable in the case of complete block and highly suggestive in the case of incomplete block, not only in determining the presence of thrombosis but also as criteria as to which side is involved.

Absence of block as determined by spinal fluid tests is also of value in the differential diagnosis of sinus thrombosis.

The test has proved of special value in cases presenting double mastoiditis developing symptoms of lateral sinus thrombosis.

While theoretic dangers must be admitted in carrying out this procedure, in 84 cases we have not seen evidence of ill effect from the technic.

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DISCUSSION

DR. H. P. MOSHER, Boston: The Doctor has covered the whole subject, so there is not much left to say that is new. We are always interested in what is new, although I shall say a word or two that is old.

Speaking of symptoms, the Doctor made the statement that there is no symptom complex in sinus thrombosis. There may not be a symptom complex in the sense that all of the symptoms are of equal value, but I have a feeling, and some of you men on the examining boards I am sure have the same feeling, that there is one symptom which stands out prominently and is of more value than the others, and that is temperature. The temperature may be of the pump handle type, or it may not be marked; there may be a slight rise, in other words, hardly more than the chilly sensations that are the results of this temperature, but frequently a temperature chart is necessary, taken as often as every two hours.

Speaking of the leucocyte count, the Doctor stated it was of little value. It may be of little value diagnostically, but I think it is of considerable value in the prognosis. In other words, whenever there is a high leucocyte count, we find the patient's resistance is slight, he is not reacting well, and it makes you feel you should hurry the operation.

Typhoid fever is a name which should be barred from an otologic clinic. The house officer who speaks the word "typhoid" immediately drops in my estimation. It is not possible for anybody over 50, who is doing otologic work, to mention the word "typhoid" in connection with ear disease.

Coming to the new part of the paper, the Abercrombie-Trendelenberg test is one of great value. Its greatest value is when you are puzzled by having some infection of the middle ear, with symptoms of lateral sinus thrombosis, and you do not know on which side to operate. It is pleasant to have it as confirmatory evidence in case of lateral sinus thrombosis on one side.

As to the technic—any test which can be put out of business by the patient grunting, or holding his breath, or sighing, is a very delicate test, to say the least. So it does have one drawback. It cannot be done unless you prepare yourself to do it. The method of doing it, I happen to know, has changed, naturally, as the test has been tried out by the originators. In the beginning they made pressure in one way, and then they gradually changed it. But to sum it up, here is a new test coming from that part of the country where we like to think new things come from—but not as often as they used to—and it is in my opinion a very valuable test.

Dr. Joseph C. Beck, Chicago: As Dr. Mosher has said, it is not always the new that is the most appreciated, but in this case I feel it is, because while most of what Dr. Tobey said, as far as he got in his paper, is a matter of repetition of what you all know, I am sure if he had finished, he would have had something to say in regard to early inteference when a diagnosis of sepsis is made—early operation.

I think, in connection with this test, Dr. Crow's name ought to be mentioned; not that he had anything to do with spinal puncture, but the compression of the jugular vein on the free side in order to see a distention of the vessels in the fundus of the eye, is supposed to be of value in diagnosis of lateral sinus thrombosis. In regard to that test, it has left me absolutely without enthusiasm after trying it a number of times. In the first case in which I employed it, the test was positive, but after that it did not show anything of value.

A matter I would like to mention, is that the test be made in borderline or positive cases due possibly to a natural narrowing on one side—real congenital anomalies, in which we occasionally find pressure. The manometrics should be brought out in such cases. If one does not, have in mind such possibilities and a positive diagnosis is made by means of this test, the case may be rushed to operation before it is ready, just like a mastoid case. Too many mastoids are operated on early—not that it hurts the patient particularly to operate on his mastoid, but the pathology of the mastoid, that is, the osteitis, is not ready for operation the first two or three days. The healing is very much more delayed than if we wait until the proper time for operation, say at least a week or two. Exceptions to this rule, of course, exist.

I do not mean to speak disparagingly of the test that Dr, Tobey has brought out; but in an audience such as this, I think it is well to call attention to the fact, that after a needle is passed into the spine and the manometer rises even to the degree which is diagnostic of sinus block, it will do no harm to wait for other corroborative symptoms.

Dr. George L. Tobey, Boston, (closing): In regard to fever, we must not forget that a great many cases of sinus thrombosis are afebrile in character. In 1918, I reported 73 cases of sinus thrombosis, 11 of them afebrile in type.

Another point, is the fact that in a great many cases of mastoid of several weeks duration, or where we have a perisinus abscess, we have demonstrated that we have a blocking of the lateral sinus giving no clinical symptoms whatever. In the first few cases we ran across, we opened the lateral sinus, proving our deductions, but since that time we have not opened them, but have gone on the assumption that the

tests are correct; and if the thrombosis was apparently sterile and not causing any sepsis, we have not interfered. We have proved, clinically, that these cases have gone on without evidence of general sepsis. So we think it is a fair statement to make, that a great many cases of perisinus abscess of long standing do have a lateral sinus thrombosis, which it is perfectly safe to leave alone.

In regard to typhoid fever, since I am under fifty, I think I may be excused.

Owing to the shortness of time, I was unable to finish my paper, but in regard to the Crow test, which Dr. Beck mentioned, we have been unable to demonstrate to ourselves any definite value in the majority of our cases.

In the question of primary thrombosis, in children especially, this test will be of great value during the course of the acute otitis media, where the patient is unquestionably septic. This test will give you data as to whether you have an infection of the jugular primarily, without infection of the mastoid. We have seen cases of this type in children and also in adults.

THE MANAGEMENT OF BUPHTHALMOS.

JAS. M. PATTON, M.D.

OMAHA, NEB.

Little Harry M., three years old, was brought to us in May, 1923, on account of poor vision. The corneae were large and hazy, the anterior chambers deep, sclerae dark, tension much increased, and vision reduced to the ability to see large objects. After doing several operations which I shall mention later, the vision is worse than when we started, although he still sees enough to get around.

A fourteen year old boy, Everett O., came to our service at the Dispensary, College of Medicine, University of Nebraska, in June, 1925, with a typical picture of buphthalmos. He had large corneae, deep anterior chambers, tension ranging up to forty-five millimeters of mercury (Schiötz), fields reduced to about ten degrees in each eye, but with a central vision of 20/70 in the right eye and 20/40 in the left. He gave the history of having had trouble with his eyes for several years, and had been wearing glasses which he recently lost, which accounted for his visit to the Dispensary. We were unable to control the tension with myotics. Repeated sclerotomies were distressingly temporary in their effect. About three months ago, filtering operations were done on each eye, and at our last examination a few days ago, his tension was twelve millimeters in the right eye, nine in the left, and there has been no further contraction of the visual fields. His vision with correction is still 20/70+1 in the right eye and 20/30+ in the left, but the time since the operation is too short to form any conclusions as to the future and the prognosis is grave to say the least. He is a bright chap and has kept his grade in school in spite of his handicap. Several cases of this character have been brought to us in the last six or eight years. As oculists we see a considerable number, and while occasionally our efforts meet with some measure of success, too often in spite of our best efforts, the vision is lost.

It is not my purpose to discuss cases of secondary glau-

coma in children, sometimes grouped with buphthalmos, or to discuss the various theories of etiology of this condition. As to diagnosis, the large cornea, deep anterior chamber, increased tension, and failing vision make up a picture that is all too familiar to the practicing ophthalmologist. We have a definite responsibility in these cases, and it has been suggested that a heart to heart discussion of our methods, a careful consideration of the procedures that give the most encouragement, might result in a higher percentage of success in the management of this trouble and thereby preserve useful vision, which otherwise is almost sure to be lost.

With this idea in mind, I made inquiry of a considerable number of the members of this society concerning buphthalmos with special reference to age, heredity, progress during observation, vision, fields, tension, nonsurgical and surgical management. Several were happy to state that they had not seen a case of buphthalmos in many years of practice, and they are to be congratulated. A surprisingly large number did not have their cases cross indexed under diseases, so were unable to trace cases which they may have had, and incidentally points a moral by which some of us, including the author, may well profit, in emphasizing the value of a carefully kept up cross index of the more grave ocular lesions that come to our attention. A good many to whom the inquiries were sent were away for the summer. Nevertheless, several excellent case reports and expressions of opinion were received which are briefly summarized as follows:

Generally speaking, there are three rather definite methods of management: 1st, the use of myotics alone, with the rather fatalistic hope that the process will come to a standstill; 2nd, the employment of one or more of the various surgical measures that sometimes seem to be of benefit but are often disappointing; and finally the combination of the two methods.

Approximately one hundred and eighty-three cases were reported as having been seen—approximately, as some of the statements were somewhat indefinite. From the reports, both eyes were usually involved, in some instances one being noticeably the worse. Many eyes were reported blind or with vision reduced to hand movements, light perception, etc., at the time when first seen. Tension ranged from plus one to plus three with the fingers, or from twenty-five to eighty-five millimeters of mercury (Schiötz). One well known observer,

reporting "about fifty cases," had not seen a tension above thirty-five (Schiötz), while another of recognized ability reported a case where the tension was eighty-five millimeters. One reported that "fields were usually fair," another that "fields were contracted," others reported vision too poor or patient too young to measure the fields. Five reported that the corneae increased in size during the period of observation. Nearly all the cases reported were under five years of age, one reporting under five, seventy-five percent; five to ten, twenty percent; over ten, five percent. Another reported ten cases all under ten years of age. A congenital mole on the forehead was the only associated anomaly reported. One case seen by us was feeble minded, and two others had peculiar shaped heads, suggesting congenital defects, and seemed subnormal mentally. No case of hereditary influence was mentioned. Dr. Peter of Philadelphia saw three cases in one family. Dr. Greene of St. Louis treated a brother and sister with this condition, and two of our cases were twin sisters.

The reports of the therapeutic management were on the whole rather surprising, forty-five percent of those replying instituting no treatment whatever for the glaucoma, enucleating the globe when pain or deformity indicated the operation. Treatment not mentioned, or nonsurgical measures were considered as good as any by twenty-seven percent. One well-known clinician and teacher, who has seen twenty cases, says, "No treatment I have tried has had any effect on preserving the vision;" and another with more than twice as many cases thinks, "nonsurgical measures as good as any, or at least until something new is found that offers better prospects." Twelve cases were reported with the statement that neither surgical nor nonsurgical treatment appeared to be of much value, while another had seen thirty-two cases but gave no hint as to what the treatment had been.

This would seem to indicate that the treatment of buphthalmos is practically nil so far as preservation of vision is concerned, and the reason is obvious. Many of the cases seen by the above seventy-three percent were blind at the first examination, and some were seen but once. We are familiar with the result of trusting to myotics alone in simple glaucoma, the "drops" often establishing false security in the mind of the patient, who continues with them until the fields and vision are hopelessly reduced. How much more so in the case

of buphthalmos, where we are told there is no anatomic basis for our hope of improvement, and opinions vary as to just how we get the benefit we do from their use.

However, there is a more encouraging side to the picture, and it is this phase which should be emphasized. Several reported cases which finally became blind, and we must expect this in a good many instances. On the other hand, judging from reports and from our own experience, there are a certain few whose vision has been kept, and this paper is a direct plea for these few.

For example, Dr. A. L. Beck, New Rochelle, New York, did iridectomies on both eyes on a ten months old child with undoubted buphthalmos, in 1913. One-half percent pilocarpin solution was also used. Ten years later, the corrected vision in the right eye was 20/70—1, in the left eye 20/100, which we must agree is infinitely better than total blindness.

Dr. Peter, reporting eight cases, has seen no improvement on nonsurgical treatment. He has employed repeated posterior sclerotomies, scleral and sclerocorneal trephining, and feels that the latter is the only method "of treatment that offers any hope of improvement." He urges that "surgery should be continued in spite of the unsatisfactory outlook in most cases. While sclerocorneal trephining with complete iridectomy has not saved many eyes for me, the period of operation being too short to give an opinion of value (three or four years), it seems to me it offers more than anything else at our disposal. It has improved vision and cleared the cornea over the period of observation." He urges the improvement of the general body resistance, and has found small doses of tincture of iodin to be of value.

Dr. John Greene, Jr., St. Louis, favors trephining plus myotics. Dr. Lancaster, Boston, believes in operative treatment and prefers the trephine. Dr. Robertson, Concordia, thinks the trephining operation of value in staying the progress of the disease. Dr. Ellett, Memphis, trephined four cases with results which on the whole were good. In one early case, the progress of the disease was arrested and vision retained, and two cases were improved. He thinks the one hope of improvement in these cases is in surgical treatment. Dr. Bedell, Albany, who has seen nine cases in the last ten years, prefers the trephine operation with myotics. A rather recent case operated by this method is doing exceptionally

well. Dr. Feingold, New Orleans, reports three cases, one being of the juvenile type. He employed the trephine operation, using the Elliott technic in the juvenile case, with an arrest of the condition when seen a year and a half later.

During the last ten years, in association with my colleagues, Drs. Harold and S. R. Gifford, we have seen thirteen cases of this condition, of which nine were definitely of the infantile type, the other four coming under the juvenile classification. Six of these were either blind at the first examination or eventually became so.

Case Reports.

C. F., female, eleven months. Right eye larger than normal, with deep anterior chamber. Fundus and tension normal. Left eye markedly buphthalmic. Cornea slightly hazy. Fundus not seen. Referred back to family oculist with suggestion to do sclerotomy followed by trephining if condition continued, rather than to do iridectomy. Unable to get further notes on case.

H. C., male, eighteen months, with typical buphthalmos. Very poor physical condition. Unable to get vision or tension. Referred to family oculist suggesting if child's general condition improved, anterior sclerotomies should be employed. He reports that he did three sclerotomies, two on the right eye and one on the left. In spite of this, the eyes continued to enlarge until about a year ago, when the right one was ruptured by a slight blow while playing. He is now six years old and has been totally blind for a number of years.

A. T., male, twenty-one months. Seen August, 1922. Consulted because the right eye was sore and watered considerably. Right eye was decidedly buphthalmic. Left eye cornea larger than normal. Right eye pupil fifteen to sixteen millimeters, left eye about thirteen. Tension right eye sixty millimeters (Schiötz), left eye thirty-seven. A broad, subconjunctival, anterior sclerotomy was done on the right eye and eserin prescribed for each. He returned a month later with the right eye free from irritation but the tension still increased. Another sclerotomy was advised but was refused. Unable to get further notations on the case.

A. N., female, three years old. Blind since infant. Mental development much impaired. Two other, normal, children in the family. Returned at the age of ten with considerable

pain and irritation. Owing to the irritation, both eyes were eviscerated. The left eye showed marked calcareous degeneration.

D. H., male, age five years. Seen in July, 1920. Vision right eye light perception questionable, left eye 20/70 questionable. The right cornea was large, sclera dark, anterior chamber deep. There was a high degree of myopia with very deep excavation. The left eye was apparently normal except for the myopia. The right eye was blind and not causing any discomfort, so no operation was advised, but eserin, one grain to the ounce, was given to use in the right eye every day, left eye on alternate days as a prophylactic. His family physician reports that the right eyeball is somewhat larger, iris tremulous, globe softer. The fellow eye is normal.

One juvenile case in this classification consulted us in 1918. W. W., male, 37 years old, had been totally blind for fifteen years, the condition in evidence at the age of twelve. He presented a typical appearance of buphthalmos. Tension was plus one to two with fingers. The corneae were about thirteen millimeters in diameter, and were slightly hazy. The anterior chamber was very deep, nerves atrophic with but slight if any excavation. There was no particular irritation. As he had no light perception, an operation was not advised but he was given a solution of eserin to use three times a day as a prophylactic.

G. W. C., male, aged four months. Seen March, 1917, with a moderate degree of buphthalmos in the left eye. The right eye was apparently normal. Right cornea eleven millimeters, left cornea thirteen to fourteen. The left cornea was hazy, tension decidedly increased. Anterior sclerotomy done on left eye by Dr. H. Gifford. Tension returned, and a month later another sclerotomy was performed, followed by a trephining just back of the limbus. There was no iris prolapse. Child seen again in August, 1917, with a questionable increase in tension. However, it seemed best to do another anterior sclerotomy. In January, 1918, the eye looked clear and any increase of tension was questionable. Nothing done. In June, 1923, the next examination, the child had a vision of 20/20 in the right eye, 20/200 in the left. Right eye, tension 17; left eye 20 (Schiötz). No increase in corneal measurements. In June, 1924; vision right eye 20/20—3 with correction, left eye 20/200 at times. Tension right eye 14 milli-

meters, left eye 17 millimeters (Schiötz). There were no fundus changes although the left pupil was still somewhat larger than the right.

E. E. T., female, age 2. One of twin girls. Eyes larger than normal at birth. Corneae noticeably hazy at six months. Both eyes markedly buphthalmic. Corneae about 14 millimeters. Anterior chambers deep. Slight photophobia. Degree of vision not determined, but mother has noticed that vision seems to be getting worse and that fields are contracted. Parents' eyes normal. No other ocular defect in family except twin sister who has same condition. Child keeps head turned down. Never looks straight at any one. Sclerotomy was advised and done a week later. Child seen again in October, when there was less irritation and she evidently had more vision as she seemed to look around more. There was a small but distinct bleb at the site of the sclerotomy. No iris prolapse. Two months later, tension in the right eye was 25, left 27 millimeters of mercury (Schiötz). Another subconjunctival anterior sclerotomy was done on each eye.

E. R. T., female, age 2. Twin sister of last case. Both eyes large at birth but no history of corneal haziness. Both children have large round heads, prominent foreheads with the suggestion of retarded mental development. Anterior sclerotomies were performed on the same date as the sister. In October there was a decided bleb at the site of each sclerotomy but no prolapse. At the December visit, the mother was sure that the child's vision had decidedly improved although degree of improvement was not determined. Tension right eye 32, left 37 millimeters of mercury (Schiötz) previous to the sclerotomy. A note from the father dated October 14, 1925, says in part, "so far as we can see their (the twins') eyes are in good condition with no change in the size of the eyeballs. E. E. is not so quick to see an object as E. R. but their eyes are clear and their vision good."

H. M., Jr., male, age 3 years. First consulted us May 15, 1923, with extensive buphthalmos each eye. Can see to get around and play with toys but vision apparently much reduced. Tension increased but not taken with tonometer Under general anesthetic, anterior sclerotomy done on each eye with induced iris prolapse. May 18, tension about 15 millimeters. There was a large subconjunctival prolapse outer side of the right eye, left eye small iris prolapse not covered

by conjunctiva. May 20, iris prolapse on the right side was punctured subconjunctivally, on the left eye the prolapse was snipped off and the opening covered by a conjunctival flap. Next seen June 27, 1923, at which time the tension of the right eye was 17, left eye 13. There was slight congestion, but the cornea was practically clear and there was evidence of edema about the paracentesis opening indicating drainage. On October 16, both eyes were free from irritation. Prolapse on the right eye is nearly ¼ inch across. Subconjunctival puncture done under general anesthetic. December 10, 1923, tension of the right eye was 34 millimeters, left eye 39 millimeters of mercury. He was not seen again until July 21, 1924, at which time there was a very large prolapse bleb the size of a small hazelnut on the right eye; on the left eye the prolapse bleb was small. Tension was 37 millimeters of mercury each eye. A double transfixation of each bleb was done under general anesthetic. There was very little reaction and the tension became normal. October 22, 1924, vision reduced but still able to see large objects. For example, could see small yellow teddy-bear toy five inches long at a distance of thirty inches with either eye. His prolapse was again the size of a small hazel nut on the right side, a portion protruding between the lids when the eye was opened but covered when the lids were closed. There was not much congestion but the light irritated his eyes. Under general anesthetic, a large conjunctival flap was raised and a section of the wall of the prolapse removed. Prolapse completely collapsed with loss of fluid, which was probably aqueous. In spite of this severe decompression, his vision was not decreased and the tension became somewhat subnormal. On April 9, 1925, he was still able to see large objects but had difficulty in seeing things close. Staphyloma had again reformed and protruded between the lids when the eyes were opened, but was completely covered by upper lid when the eyes were closed. Protecting goggles of laminated glass were prescribed. As the resections of the staphyloma had not been of much value, at the suggestion of Dr. Harold Gifford, a trephining was done at this time with a 1½ millimeter blade. Iris prolapsed into the opening, and a small iridotomy was made, followed by a gush of fluid, possibly aqueous, but it might have been fluid vitreous. The conjunctival flap was replaced and held in place with sutures. There was slight bleeding from the iris into the anterior

chamber. At the end of a week, the eye was free from inflammation, with the vision about the same as before the operation, and he was sent home. About two months later, a long distance phone message from the family physician stated that the eye had become suddenly inflamed with evidence of deep infection. Inflammation continued a few days, when the eye suddenly collapsed following a gush of fluid, and it seemed as though the sight would certainly be lost. Palliative treatment was advised, and nothing further was heard of the case until October 7, 1925, when his family physician, Dr. James McAllister of Odebolt, Iowa, writes, "The inflammatory condition finally subsided. The anterior chamber began to clear up but the eyeball was very soft, the staphyloma having almost disappeared. The vision gradually improved, but the tension increased almost in proportion. Today his mother thinks he sees almost as well as before the last inflammatory reaction, but I do not think so. I do not think the tension is quite up to normal. The staphyloma is very slowly becoming more prominent. The conjunctiva is clear and his general condition is very good. His mother says that the child sees distant objects when outdoors rather than close objects. While in my office, he spied a stamp on an envelope and called my attention to it, and also recognized the red color at a distance of about eight inches. The eye looks better than it did six months ago, but I do not think the vision is as good as it was then."

A. H., female, age 11. Seen in November 1920. High myopia. Vision with correction 20/50 each eye. Three other children in family. No evidence of this trouble. No history of trouble with parents. Both corneae very large, anterior chamber deep. Fields moderately contracted. Right eye has deep excavation of disc, left eye excavation not so pronounced. She was returned to her family oculist with suggestion to do repeated subconjunctival anterior sclerotomies, which was carried out. In June, 1921, the vision was the same. Tension was 28 millimeters of mercury (Schiötz) each eye. In July, 1923, vision of the right eye was hand movements, left eye 20/50. The right cornea was slightly hazy, iris tremulous. No discomfort noticed. An iridotasis was done on the right eye and a broad anterior sclerotomy on the left by Dr. Gifford. She went home without unusual reaction, but in August her family oculist wrote she had considerable pain in the right eye

and was advised to use massage on the globe. A week later, she was free from pain but without improvement in vision. They moved to the west coast shortly after this, and in March, 1924, the mother wrote that the right eye was still irritable. A report in October, 1925, states that the right eye is totally blind, the left one "holding its own."

E. O., male, 14 years old. First seen on my service at the Dispensary of the College of Medicine, University of Nebraska, June 13, 1925. He had been wearing glasses for two or three years, but had lost them a month previous and came for refraction. On examination, the pupils were found to be extremely dilated, the conjunctivae slightly inflamed, with vision of 20/100 in the right eye, 20/50 in the left. The ophthalmoscope showed light discs with considerable excavation. The blood vessels were somewhat engorged and with a tension of 45 millimeters of mercury (Schiötz) in each eye. Eserin solution, one grain to the ounce, was prescribed to be used four times daily. Although his central vision was fairly good, his visual fields were contracted down to about ten degrees in both eyes, giving him a narrow tubular field. As he also had enlarged tonsils and considerable adenoids, these were removed by the laryngologic staff June 29, 1925, without any effect one way or the other on the condition of his eyes. He was seen twice a week at the Dispensary with treatment, test of vision, tension and fields. The vision and fields remained about the same, but the tension fluctuated from 20 to 25 millimeters of mercury (Schiötz) at the lowest, up to 37 to 40 at the highest point. As the myotics were evidently not controlling the situation, on July 22, he was sent to the hospital, where a double anterior sclerotomy was done under general anesthesia. This was only temporary in its effect. The frequency of the myotic was increased to six times a day, but on August 8, 1925, tension had risen to 45 millimeters of mercury (Schiötz) each eye. The margin of safety for his vision was so narrow it seemed advisable to do something in the way of a filtering operation, so he was taken immediately to the hospital and a double iridotasis was done by my associate, Dr. S. R. Gifford. There was very little reaction, but to be on the safe side myotics were continued, and an examination early in September, 1925, showed the tension of the right eye to be 20 millimeters, left 23, reducing to 15 under the tonometer. Vision in the right eye was 20/70, left eye

20/40—2, improving to 20/30 with correction. A slit lamp examination at this time showed no rupture in the membrane of Descemet. The fields were still contracted down to about 10 degrees, with some central blurring. On examination October 10, 1925, with correction his vision is 20/70+1 in the right eye, 20/30 in the left. The tension is 12 millimeters in the right eye, 9 millimeters in the left (Schiötz). The anterior chambers are deep, pupils not particularly dilated. In the right eye, there is a moderately well marked staphyloma in the region of the iridotasis bleb. In the left eye, the staphyloma is very small. Both eyes seem to be draining satisfactorily as indicated by the reduced tension.

W. O. C., male, age 15, vision corrected to 20/30+1 and 20/20—. Consulted us in May, 1922, on account of a divergent strabismus. Corneae were large, anterior chambers deep, but pupils reacted promptly to eserin. Both discs were very light and deeply excavated. There was almost a complete horizontal hemianopsia in the right eye, the upper field being absent. There was moderate concentric contraction of the left field. Tension in the right eye was 65, left 70 millimeters of mercury. A large subconjunctival sclerotomy was done on the right eye. Eserin was prescribed and the tension came down to 25 and 23 millimeters of mercury respectively, but a week later was 37 in the right and 33 in the left. Repeated anterior sclerotomies were done on both eyes, with fluctuation of tension up to 35 or 40 millimeters until January, 1923, when an iridotasis was done on the right eye and a peripheral iridotomy on the left. There was no special reaction, but as the iridotasis bleb showed a tendency to increase in size, a subconjunctival puncture of the bleb was done about the first of March. This was followed by diffuse edema of the conjunctiva in the region of the bleb, which is present to date. At the last examination, October 1925, tension in the right eye was 12 millimeters, left eye 32 millimeters (Schiötz). Vision was 20/40 and 20/20 with correction. Fields slightly better than at first examination three years ago. He has continued the use of eserin, but in spite of the fact that his vision and fields have not changed for the worse, it would seem advisable in view of the increased tension to attempt to establish permanent filtration in the left eye.

A hasty review of the recent literature does not present a very encouraging picture, but as in the report given above,

sufficient improvement is noted to justify us in giving the patient the benefit of such means as are at our disposal. Cucco[1] favors early trephining, but visual results are not given in the abstract. Harlan[2] presented a case in which there was no change following iridectomy. H. Gifford[3] reported two cases trephined without improvement. Zentmayer[4] reviewing the status of therapeutic management up to that time, noted that "Limitation of the progress of the process is not, however, consistent enough to justify tentative treatment." He favored iridectomy or sclerectomy, felt that the trephine operation was attended with the least hazard, and that the only method giving encouragement was some form of sclerectomy. Fox, in discussion, favored sclerectomy but reported no improvement in cases operated. Snell[5], writing of juvenile glaucoma, has found that iridectomy is of little value and thinks the trephine is the operation of choice. In discussing a case of buphthalmos presented before the Colorado Ophthalmological Society in February, 1922, by the doctors Bane[6], who had been using myotics without improvement and were considering trephining, Walker, favored operative measures, having obtained encouraging results in such a case, while Black, Crisp, and Coover rather favored the continuation of the myotics. De Lord[7] reports a case of one sided buphthalmos treated by two sclerotomies, in which the tension is normal six months after the last operation. Goerlitz[8] trephined four eyes in three severe cases, which have been under observation for two, three, and four years respectively. In each instance function returned, the tension was reduced, excavation of disc and progressive enlargement of globe stopped. No filtration scar was evident, the wound healing flat. He considers trephining safer than iridectomy.

Poulard and Lazart[9] followed eleven cases from 1913 to 1924. Twenty operations were done, sixteen with the trephine. In twelve of the seventeen eyes, the tension was still below twenty-eight millimeters of mercury (Schiötz). One case after twelve years had a vision of one-fourth in each eye, while six have sight permitting of normal activities. In three cases there was no improvement.

Blake[10] reviews the literature and reports on the observation of 472 cases of hydrophthalmos with 156 operations. No summary is given as to visual results, but he mentions several cases, either from his reports or from the literature, where vision was retained or improved by the combined method of

treatment. He emphasizes the importance of treatment during the first year of life, if seen that early, and favors the use of myotics before and after operation. A combined trephine and iridectomy is in his opinion the safest, and on the whole offers more than any other operative procedure under consideration.

Summary.

Unfortunately, a large percentage of the cases of buphthalmos seen by us are blind at the first examination, and offer no hope of improvement no matter what the treatment may be. However, all cases with useful vision, and infants with even light perception, should be given the benefit of operative measures. Cases of spontaneous arrest of the disease are exceedingly rare, and no case was brought to my attention where myotics alone were successful in preserving vision, while surgery or surgery plus myotics have saved a considerable number of individuals from total blindness, with useful vision in many instances.

Some seem to take the position, that as the prospect of a successful outcome is so poor, why subject the patient to a rather serious operation, or risk our professional reputation with such doubtful hope of improvement. Neither attitude is worthy of our profession. Even if we were able to save only one in a hundred (and the results of surgical intervention are much higher than that, ranging from twenty to fifty percent with many operators), it would be worth the risk. We can not tell which cases will be successful and which will fail, so we insist that cases which offer any hope at all should be given their chance. As to the type of operation, the personal preference of the operator must largely determine which he will select. Subconjunctival, anterior sclerotomy is the easiest, but must be frequently repeated and usually does not give permanent reduction of tension. Sclerotomy with spontaneous or induced iris prolapse reduces the tension, but the prolapse tends to increase in size and is difficult to manage. Iridotasis, in three eyes operated by us, has given satisfactory result in the short time under observation. The classical iridectomy, while favored by a few, is generally considered to present unwarranted risk due to the relatively large incision. Generally speaking, the best end results have been reported

by men who favor some form of sclerectomy, preferably the sclerocorneal trephine operation, or a scleral trephining immediately back of the limbus. Do not be satisfied with one operation if the tension is not permanently reduced. Repeat the procedure, and do not give up so long as there is any vision worth saving.

Myotics should be used for at least a year after operation, or indefinitely if there is any question about the result. Dr. H. Gifford insists on the use of a mild collyrium, preferably a 2/10 percent solution of zinc clorid in a supersaturated boric solution, instilled twice daily as a prophylaxis against secondary infection.

We must remember that the same rule applies as in other forms of glaucoma, namely, that sudden reduction of high tension may be disastrous. If a sclerectomy or other extensive operation is planned, first reduce the tension slowly by a subconjunctival limbus puncture.

In conclusion, let us remember that every case of buphthalmos is not hopeless, that intelligent persistent surgery, with myotics, will save a few at least, and that we, as ophthalmic surgeons, are personally responsible to these few to do for them all that is possible.

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DISCUSSION.

DR. J. L. MYERS, Kansas City, Missouri: I am very glad that Dr. Patton has given the word of encouragement he has in the treatment of buphthalmos. I want to show some pictures of patients in the Blind School, Kansas City, Kansas, which I am able to show through the courtesy of Drs. May and Dwyer, who have charge of that work. These patients have come from various parts of the country, and they show just how the medical profession as a whole is looking on buphthalmos. As you look at these pictures and see the condition of these patients, you are made to wish that some interest could be aroused in the profession that would cause more work to be done, and from the splendid reports that Dr. Patton has given, we are forced to the conclusion that we are justified in spending much more time in studying the pathology of this condition, if by so doing we can give sight to even a small number.

SOME CAUSES GIVEN FOR BUPHTHALMOS AND GLAUCOMA

1. CONGENITAL SYPHILIS.
2. SPECIFIC CHOROIDO-RETINITIS.
3. INTRA-UTERINE INFLAMMATION.
4. HYPERSECRETION OF VASO-MOTOR ORIGIN.
5. HEREDITY.
6. CONSANGUINITY.
7. ARRESTED DEVELOPMENT OF FILTRATION ANGLE.
8. ABSENCE OF SCHLMM'S CANAL IN 50% OF CASES. (HAMBERGER)
9. DISTURBANCE OF ENDOCRINE SYSTEM.
10. UNEQUAL SWELLING OF COLLOIDS OF THE EYE. (FISCHER).

Fig. 1.

We have gone over the literature to find the causes given for buphthalmos and glaucoma, and find them to be:

First, congenital syphilis; second, specific choroidoretinitis; third, intrauterine inflammations; fourth, hypersecretion of vasomotor origin; fifth, heredity; sixth, consanguinity; seventh, arrested development of filtration angle; eighth, absence of Schlemm's canal in 50 per cent of the cases; ninth, disturbance of the endocrine system; and tenth, unequal swelling of colloids of the eye.

The next slide gives the various methods of treatment that have been used, which are: miotics, iridectomy, trephining, sclerotomy and subconjunctival injection of sodium citrat solution (4.05 per cent to 6.41 per cent., 15 to 20 gtts.)

These pictures show the tragic end of some cases of buphthalmos.

NAME	SEX	AGE	VISION	B.W.	TREATMENT
L.W.	M	12	OBJECTS	4 plus	ESERINE
M.O.	F	10	R.Light L.4/200	Neg	NONE
E.K.(col)	F	20	0 10/70	Neg	NONE
I.L.	F	20	OBJECTS	4 plus	ESERINE. K.I.
L.S.	F	24	NONE		NONE

Fig. 2.

and some which we hope will stimulate a deeper interest in finding some method of giving relief.

The first case is a boy, twelve years of age, who had scant vision ot movement of the hand. This patient's Wassermann was plus 4. The only treatment he had ever had was eserin.

Fig. 3.

Fig. 4.

The second case is a girl, twenty years old; right eye, no vision whatever; left eye, 10/70; Wassermann negative; no treatment whatever.

The third case is a girl, twenty years old; had vision in one eye only and could see objects only; Wassermann plus 4. The only treatment was eserin, dionin and potassium iodid.

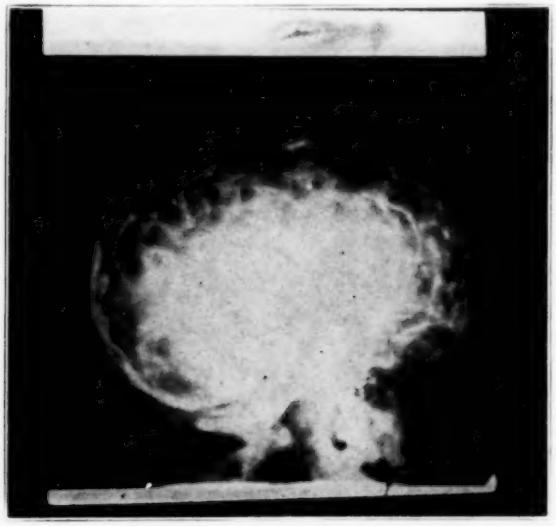


Fig. 5.
Roentgenogram showing tower skull, Fig. 4.

Fig. 6.

The fourth case is a girl, 24 years of age, no vision whatever; Wassermann negative: no treatment.

The fifth case is a girl, 10 years old; right eye could see light only; left 4/200; Wassermann negative; no treatment.

It is so frequently stated that cases of buphthalmos have congenital

Fig. 7.

Fig. 8.

syphilis, and yet in the tests we made of these five, only two gave a positive reaction. Another thing that is brought out in these cases, is that very little, or nothing, has been attempted for them. The question is, what are we going to do? Our tests of the blood have shown that in each case, throughout, the blood was negative. So far as we have been able to learn, the treatment of those cases where it was thought that the condition was due to swelling of the colloids of the eye, have proven of no more effect than the use of eserin, dionin, potassium iodid, and so forth. The thing we must do is to depend on surgery until we get some more information on these cases. Because we have been unsuccessful in

Blood Chemistry in Buphthalmos

Case	Urea Nit. Mg %	Sugar Mg %	Chlorides as NaCl Mg %	Creatinine Mg %	N.P.N.
1	10.9	97	475	1.3	21.7
2	7.2	106	466	1.5	17.1
3	9.8	102	458	1.7	20.0
4	11.2	102	491	1.5	—
5	8.7	95	450	—	16.9
	9-14	80-120	[illegible]	1-2	

Fig. 9.

TREATMENT OF BUPHTHALMOS
AND
GLAUCOMA

1. MIOTICS
2. IRIDECTOMY
3. TREPHINING.
4. SCLEROTOMY.
5. SUBCONJUNCTIVAL INJECTION OF SODIUM CITRATE SOLUTION (4.05%-to6.41%) 15 to 20 gtts.

Fig. 10.

treating them so far is no reason why we should be discouraged; it is all the more reason why we should try harder to find the conditions causing the trouble, and then try to find something that will give relief. It is not right that we medical men should do nothing for them.

Dr. Daniel W. White, Tulsa, Oklahoma: I want to offer a few words on my experience with the spinal fluid in these cases. From the objective and subjective symptoms in four cases, a diagnosis of probable glaucoma was made. The spinal fluid pressure in the first case was 20 mm.; in the second, 14 mm.; third, 6 mm., and the fourth, 10 mm. The amount of spinal fluid withdrawn was in the first case, 3 c.c.; second case, 2 c.c.; third case, 2 c.c., while in the fourth case the spinal fluid was not withdrawn. After treatment by mouth with salicylat of soda, thyroid

extract, epsom salts baths, etc., the vision showed 15 per cent improvement in the first case, 2 per cent in the second, and no improvement in the last two cases.

The question has come to my mind, is this condition a slow myelitis, or is it an indirect disturbance of the aqueous fluid, or is it a disturbance or the spinal fluid without a myelitis, or is it a slow cerebrospinal fluid pressure uveitis? I feel the spinal fluid should be removed in every case as soon as you see it, and a bacteriologic and pathologic study made. I also feel we should investigate this field more thoroughly, to see if we cannot clear up some of those conditions.

Dr. James M. Patton, Omaha, Neb. (closing): I want to reemphasize Dr. Meyers' charts. There were five cases rendered blind, 100 per cent loss, and only one of them had had any surgical treatment. We must get away from the idea that buphthalmos is 100 per cent loss. There are many cases reported where useful vision has been kept after operative measures, and through the use of operative measures plus miotics. I think we are guilty of gross negligence if we do not give the cases the benefit of what we can do for them. Our operative measures are not 100 per cent good—they are sometimes not any good—but if some of these cases can be helped, we certainly are neglecting our duty if we do not give them the benefit of what has been found useful in the hands of other operators

SOME OF THE PROBLEMS OF THE HARD OF HEARING AND THEIR SOLUTION.

HAROLD HAYS, M.D., F.A.C.S.

NEW YORK CITY.

A subject of this kind is so large, that it would be impossible to enter into a general discussion at this time. Within the past two decades, great strides have been made in the management of the deaf, but I regret to say that we are far from that millenium where we can promise that we can cure deafness. This is not due entirely to a lack of desire on the part of medical men, but is due to a great extent, to two factors—first, the negligence of the patient, and second, the impossibility of definitely interpreting the pathologic condition behind the ear defect. For example, a patient presents himself with a hearing defect which shows plainly that the internal ear is involved. The question arises, is there not some middle ear condition which is aggravating the internal ear trouble? Is it not possible that the middle ear condition can be improved and even arrested? And is it not utterly wrong to state to such a patient that he cannot be improved because an internal ear pathology has been found?

The greatest encouragement has been found in the interpretation of the causes of deafness and in the management of the average case which is amenable to treatment. The time worn use of the inflation method by the catheter or by Politzerization has practically been discarded, and the intelligent otologist of today will not be satisfied until he has made a satisfactory hearing test, has examined into the causes of the ear condition, and has treated the pathology as he has found it. He has been greatly aided by having at his disposal various electric devices such as the nasopharyngoscope and the auroscope. He is now able to examine the pathology of the nasopharynx with as much exactitude as he is able to examine the oral cavity, and he is also able to interpret the conditions of the drum membrane so that he is not content with a hasty examination and the assertion that the drum is

retracted. Even in cases in which there seems little doubt that there is a nerve lesion, I have seen the hearing improve fifty percent by proper treatment of the tubal mucosa and by establishing the patency of the Eustachian tube. Another factor of great importance, which was too long unrecognized, was the effect of various general abnormalities on the hearing mechanism. The otologist of today realizes the importance of proper inquiry into various ailments which are far removed from the ears and, in the majority of instances, he will refuse local treatment unless the patient receives the proper care of his other infirmities. I have seen many a case of deafness improve after a gastrointestinal condition was cleared up, and I have known of numerous instances where the patient's hearing defect improved considerably after the general resistance was brought up to par.

But, to my mind, the one factor which will count for more than anything else in the advancement of our knowledge of this subject, is the awakening of interest in it on the part of the otologists. I can well recall the hopeless position that most of us were in until recent years. The deaf patient was considered more or less of a nuisance—at least, one felt that very little could be done for him if he did not improve after one or two inflations with the catheter. It was seldom that the otologist took the time and the pains to explain the condition to the patient, and to put him in the way of making his life more useful and happy. One often heard the bromidic remark, "I fear your condition is progressive and little can be done for you." This often meant mental suicide to the patient. Or again, "You have otosclerosis." Never was a term more loosely used. I believe that Dr. Pierce once made the remark, that we have yet to see the first case of otosclerosis in this country which was proven to be such on autopsy. Sadder than this is the fact that many a case which was diagnosed as otosclerosis on hasty examination, responded to treatment at the hands of another otologist. I have seen many a case of relaxed ear drums or pocket handkerchief deafness which was wrongly diagnosed as otosclerosis.

Conjecture as we may, there is no doubt that the psychologic state of the patient, at any given time, has a great deal to do with the ability to hear. Given an individual patient, he will invariably hear better when he is in an exalted state of happiness than he will when he is in a state of depression.

I have in mind the case of a young woman who has been a patient of mine for many years. There is no doubt that she has a nerve deafness which is aggravated by changes which take place in her middle ear. When Miss A. first came to me, it was impossible to obtain any improvement in her hearing. She was studying hard to become a teacher, desired to mask her hearing defect, and was in a continuous state of mental and physical depression. Her Eustachian tubes were well open, and no definite pathology could be found in her nose or throat. After a number of years, she succeeded in getting herself placed as a teacher, her home surroundings improved, she became more mentally alert, and she adopted a different attitude toward her hearing defect, with the result that her deafness remained stationary. Last Spring, her hearing was tested on the audiometer, which disclosed a defect of fifty percent in her right ear and over forty percent in her left ear. She left town for the Summer. A test of her hearing was made at the offices of the Western Electric Company, and in our office on one of their audiometers, and an improvement of over ten percent was found in both ears. No treatment had been given her in the interim, but her general health was improved. This is only one of many instances where the proper handling of the patient, from the psychologic point of view, has meant an improvement in hearing.

Unfortunately, as our knowledge of the pathology of deafness increases and our ability to help the deaf in various ways becomes more evident, we find that the general deaf public becomes more gullible. The main reason for this is that the treatment of the deaf has become more popular, and the charlatan has become more intelligent in his means of approaching the deaf. It is seldom that he attempts to use time worn instruments which will be a cureall. Today he adopts the socalled scientific method and poses as a scientist. He may be found among the ranks of the "outsiders," among those who suddenly conceive a form of treatment which would be farcical if it were not that so many hopelessly deaf people are fooled. He may be found in the ranks of the pseudo-scientists, chiropractors, naturopaths, osteopaths, christian scientists, etc., and more unfortunately still, he may be found in the ranks of the ethical medical profession. It is not my purpose to dwell upon any of these special methods of treatment, or to attempt to expose any of them. But our more

definite interpretation of hearing defects, as was previously pointed out, has shown us that this infirmity is caused, or at least the infirmity increased, by a multiplicity of conditions, many of which are local and many of which are general.

One may consider that the charlatan is the man who refuses to allow his type of treatment to be tested out by other scientific men, so that it may have the stamp of their approval.

The X-ray treatment of deafness has been given a fair trial, and I do not hestitate to say has not met with any marked success. Finger manipulation may have its place in the osteopathic armamentarium, but again I do not hesitate to say that it has not met with great success, except in the hands of those who are born with special fingers and the mind to guide the finger into the right places. Nasal operations, of one kind or another, have been advocated as a means of improving the hearing ever since the otologist has been able to wield a knife within the nose. Evidence points to the fact that any such improvement is transitory, seldom permanent. Theorize as we may, we are not able to overcome definite connective tissue changes by operating upon an extraneous part. The massaging of a spine may stimulate the nerve endings, but it is more likely to stimulate suggestively the digestion and the cerebral cortex, so that a person changes his thinking mechanism. The same may be said of psychoanalysis and christian science. Fortunately, the advocacy of wearing artificial ear drums for every form of deafness is going out of vogue.

To this list of special types of treatment which have been much heralded, may be added the claims of those who advocate the use of certain apparatus, such as the numerous types of vibrators, the concussors, and various forms of electric treatment, such as ultraviolet rays and diathermia. I do not by any means wish to intimate that these types of treatment (including those which are most universally condemned) have no special uses, and I agree that, in certain instances, marked improvement may be obtained, but one must insist that that one type of treatment must not be the only one used. I am a firm believer in the various forms of electro- and mechanotherapy, but I have yet to see one single case in which improvement took place by the application of any special method of treatment.

The reason for the charlatan's success is almost self evident; the removal of the evil is not so simple. His success

depends first on the gullibility of the human animal, and secondly on the intense desire of the deaf individual to rid himself of an infirmity which he at all times wishes to hide. He always feels that there is someone in this world who ought to help him, and having met with discouragement at the hands of the scientific otologists, he feels that perhaps some new man who promises much will be able to do more than the man who has spent all of his life in trying to solve this problem. No more illuminating evidence on this subject could be presented than that given by Dr. Harold Rypins, in his classic paper before the American Federation of Organizations for the Hard of Hearing, in Minneapolis last June.

Dr. Rypins' suggestion for a cure is education of the deaf people themselves. He wishes to have anyone who claims a cure for deafness brought before a tribunal—that is, before the various bodies of the deaf who are working for the deaf. He wishes an index kept on each and every form of treatment, and confessions of deaf people so treated kept on record. If there is anything in any form of treatment, no matter by whom advocated, we want to know it. If there is nothing in their forms of treatment, we want the deaf to know it. The medical men can rail against these pseudoscientists forever and anon; it will do little good. But once the deaf themselves refuse to go to these individuals, once they spread the nakedness of the truth about, then will these individuals die of starvation or try their misguided intellects in other fields.

In the not far distant past, the handling of the deaf patient was left entirely in the hands of the physician. The awakening began with the formation of various organizations of the deaf. This has meant far more than we can imagine at the present day. I am happy in the feeling that I organized the first League for the Hard of Hearing in this country; but if it had not been for the devoted interest of the late Edward Bartlett Nitchie and the Alumni Association of the New York School for Lip Reading, it would have been impossible to have obtained the wholehearted cooperation so necessary in a movement of this kind. The prime object of the New York League was social service among the deafened. No sooner had we begun this work, than we found that this was but a small branch of it. Numerous people needed instruction in lip reading; many of them had to be mentally rehabilitated; some of them needed financial aid. As soon as we began to work

on these problems, we found that we needed a consulting board of otologists, who would be able to separate the deaf people who could be helped by medical means from those who could not. And finally, within recent years, we have launched into the field of prevention and have begun to work on the children at an age when they can be improved and sometimes cured. Our figures, after working on hundreds of school children during the past few years, agree with those of others and, briefly, we have come to the following conclusions: 1, that about three percent of school children are suffering from ear conditions which interfere with their school work; 2, that the cases equally divide themselves between those who are suffering from suppurative conditions and those who are suffering from socalled catarrhal otitis; 3, that many of these children have not had their tonsils and adenoids removed, or inflammatory or suppurative conditions of the nose attended to; 4, that many of these children have a tendency toward a relaxed ear drum because they do not blow the nose properly; 5, that a majority of the suppurations can be arrested by proper treatment and without performing a radical mastoid operation; 6, that 80% of the children who are suffering from catarrhal conditions can be improved or cured by simple Politzerization. These figures are encouraging and demonstrate that it is most necessary to resort to a campaign of education until it becomes the rule for every school child to have his hearing tested at regular intervals.

About five years ago, the American Federation of Organizations for the Hard of Hearing was formed with Dr. Wendell C. Phillips, President Elect of the American Medical Association, as the first President. Mainly through his efforts and the efforts of the loyal members of various local organizations, we were launched into almost immediate success. I had the pleasure of serving as the second President, to be succeeded by that most conscientious otologist, Dr. Gordon Berry, who has placed the organization on a true scientific basis. We are fortunate in having as our President Elect the President of our Academy, Dr. Horace Newhart, who is sure to carry the work on toward its ultimate success. Both Dr. Phillips and I are proud of the fact that we have been succeeded by men who have the ambition, the energy and the enthusiasm to put over any project in which they are interested.

Why should the Federation receive the support of the

medical profession? Why should every otologist know of its work? In what way has the Federation been of help to the otologist? The reason why we, as medical men, should support it is because it is doing its utmost to advance the welfare of the deaf, medically, mentally, economically and sociologically. Every otologist should know of its work, because the Association is trying in every way to simplify the mental and physical condition of the deaf, thus making the work of the otologist much easier. In many other ways has the Federation been of help to the otologist. First, it has attempted to steer the deaf away from the charlatans. Secondly, it is collecting statistics to show that deafness is preventable, so that the deaf child will be brought to the specialist at an earlier age. Thirdly, it has compiled for you the names and addresses of the lip reading teachers throughout the country, so that you will know to whom to refer your patients. Fourth, it is now attempting to inform you as to the worth of the various hearing devices being advertised. Fifth, it is attempting to standardize hearing tests, so that all of us will make tests in the same way, and last, it has formed a general scientific committee composed of physicians, physicists, educators, etc., who, no doubt, will present some amazing data to you in the near future. Moreover, the Federation has been instrumental in the formation of committees dealing with the welfare of the deaf in our various national societies, with the result that the work of the Federation has been indorsed by the House of Delegates of the American Medical Association. Surely such work should meet with your approval and indorsement, and it should be the ambition of every otologist present to associate himself with one of the local branches of the Federation.

Summing up the advances that have been made in the treatment of the deaf, we may conclude as follows:

1. The attitude of the otologist toward his deaf patient has changed.
2. More interest is taken in the prevention of deafness.
3. Psychologic treatment is of the utmost importance.
4. The elimination of the charlatan will depend on the success of a campaign of education against him.
5. The lay organizations for the deaf, such as the American Federation of Organizations for the Hard of Hearing are helping a great deal in solving the problem of the deaf.

A DEMONSTRATION OF MODERN METHODS IN THE TRAINING OF THE DEAF CHILD WITH TEACHERS AND PUPILS FROM CENTRAL INSTITUE FOR THE DEAF.

M. A. GOLDSTEIN, M.D.

ST. LOUIS, MISSOURI.

Thirty years ago, when the American Academy of Ophthalmology and Oto-Laryngology was in its infancy and still known as the Western Eye, Ear, Nose and Throat Society, I demonstrated a class of fifteen pupils in acoustic stimulation before this society at its annual meeting in St. Louis. Three similar demonstrations have been given before this organization since, to emphasize the progress of this work with the deaf child.

The work which I have been invited to present for your consideration tonight deals more especially with the deaf child —the child congenitally deaf or the child who has acquired deafness at so early an age, that this handicap requires an unusual form of scientific consideration and of special pedagogy. Unfortunately, scientific research in the causes of congenital deafness has not yet produced sufficient data for our guidance, and progress in the education of the deaf child has made less important strides than similar progress in the uplift of other types of the handicapped child. The crippled child, the blind child and the mentally defective child have had a very fair share of consideration pedagogically, scientifically and sociologically; but the deaf child, because he has been less seriously studied and less clearly understood, has always been a neglected entity, and the pedagogy of the deaf child has been in a state of partial inanition.

When I returned from postgraduate work abroad some thirty years ago, I looked for an opportunity to apply some of the thoughts gathered in Vienna and elsewhere, so that I might check up by my own findings the principle of auditory stimulation; but there is always difficulty to find an entering wedge in the application of modern or radical ideas in any

community that is fairly well saturated with orthodoxy. Schools for the deaf in America thirty years ago had not advanced in pedagogic liberalism enough to give so new a thing as auditory stimulation an unbiased consideration. To make possible a more progressive form of training for the deaf child, therefore, I was impelled and compelled to organize an independent school for the deaf, not because I desired to place another school for the deaf in the field, but because I craved the opportunity to correlate the experience of the trained teacher of the deaf and the scientific background of the otologist and laryngologist—hence the origin of Central Institute for the Deaf in St. Louis. It was the first school to be organized and adopt as its working principle the daily cooperation of the trained teacher of the deaf and the trained otologist, and had for its goal the improvement in pedagogy and the more serious study of the deaf child. The product of twelve years of serious application of these ideas and principles is crystallized in the practical demonstration offered you tonight. As an end result of these observations, we are prepared to offer for your critical consideration a modern form of pedagogy having as its goal the emancipation of the congenitally deaf child, who, notwithstanding his serious handicap, is enabled to hold its place in the world of speaking and hearing people, to express his thoughts in our own language and in a speech that is flexible, fluent and understandable, and with an efficiency in lip reading that makes his contact with speaking humans almost normal.

In an open meeting of a scientific body such as this, where the interested laity is invited to participate, there is much opportunity for legitimate publicity. I believe that the great American public, through its greatest source of information, the American daily press and the American periodical, is entitled to all the intelligent information that may be gathered and presented concerning the development of educational opportunities and newer scientific work; concerning the publication of all principles of mechanics, such as the radio and the telephone, that might be of benefit to the deaf child; concerning the medical research that has tested out and given its stamp of approval to the application of such principles. I believe emphatically, that all such information should be intelligently disseminated; that there should be a general protest against "sob stuff" stories that are constructed of fairy tales

instead of facts, thus bringing about much unfortunate misinformation to be absorbed by an unsuspecting and innocent public. If the public at large could obtain honest and accurate information on such a subject matter, the poor deaf man who went up in an aeroplane with a pilot a few weeks ago with the idea that when he descended from the upper air strata his hearing would be restored and he would be able to speak, might have avoided death by an overzealous enthusiasm created by newspaper reports.

Think of the blasted hopes of many deafened people who are told in most glowing terms in the daily press and elsewhere of the wonderful new discoveries by which their hearing will be restored, and by which they will promptly be lifted out of the depth of despair into a hearing world. These wonder working cures must be stripped of the colorful imagination of newspaper reporters, ambitious tradesmen and quacks.

It is an unfortunate fact that there may be many opportunities vouchsafed to the deaf by the application of important principles in modern mechanics and physics such as the radio and the telephone; the application of these modern ideas have their definite scientific and utilitarian value, but they are so garbled and so exaggerated in their presentation to the public that whatever scientific and utilitarian value they possess is thus lost.

The congenitally deaf child is the principle subject for our consideration tonight. I differentiate between biologic congenital deafness and pathologic congenital deafness. In biologic congenital deafness, there may be absence or malformation of any part of the auditory apparatus in its central or peripheral aspects; in pathologic congenital deafness, there is usually a background of hereditary syphilis, rickets, tuberculosis or some intrauterine or other constitutional entity to which the deafness may be traced.

It is an interesting observation that the biologically congenital deaf child is very rarely totally deaf. This fact has been substantiated not only by the observation of investigators in otology, but by experienced teachers interested in the training of the deaf child. It is a significant fact that over thirty per cent of all pupils in all schools for the deaf in this country have some residuum of hearing. This residuum may vary from an inability to hear more than imperfect sounds of

the human voice to that of being able to interpret fluent speech at close range.

Until recently, most schools for the deaf have not seriously considered these biologically congenital deaf children with varying degrees of remnants of hearing. In many instances, they may still be found in schools where the sign language is still prevalent.

The somewhat painful controversy between the advocates of speech methods on the one hand and the orthodox adherents of the sign language and finger spelling methods on the other is hardly pertinent to our presentation. I cannot forego the criticism, however, that it is a serious error to consign to a school in which the sign language is taught a deaf child that can still hear the human speaking voice at a distance of four to five feet, and to still subject this child to instruction by sign methods. A deaf child with this remnant of hearing who is not given an opportunity for stimulation of his auditory mechanism, or where no attempt is made to reach his brain through his ear instead of by signs or mechanical speech, is unjustly dealt with. In short, such an act is an educational crime.

Our mission here is to advocate the improvement of conditions in the status of the deaf child throughout the nation; we are not here to demonstrate exclusively what Central Institute for the Deaf can do, but rather to spread the gospel of better speech, of better training for deaf children, of better opportunities, of developing teachers with better qualifications, in short, for the consideration of the improved sociologic status of the American deaf child of the future.

We have selected from our group of pupils at Central Institute for the Deaf in St. Louis examples to demonstrate various phases of the work in which an up-to-date school for the deaf is engaged, and we wish to submit these living documents-in-evidence for your impartial judgment.

Before introducing to you the first steps in the training of the deaf child, let me assume that you have never been inside of a school for the deaf, and that you will now be shown the first contact between the deaf child and his teacher.

The congenitally deaf child cannot be approached by the otologist in exactly the same manner as the deafened child or the deafened adult. The means of contact are different. The deaf child who has never heard speech and who has never

had any previous training, cannot be mentally reached by the otologist or teacher by the same means as the hearing child is reached. The otologist is often asked by the parent of a deaf child: "Doctor, please advise me whether my child is deaf and what to do for him." Voice tests are unfamiliar sounds to such a child; the tuning forks he does not know; loud noises may be simple tactile impressions. I have often spent a period of two to three weeks, including five or six sessions of one-half hour each, trying to determine what, if any, remnant of hearing a congenitally deaf child still possesses. This is an interesting field of study and research about which much can be said on another occasion.

(Miss May Templeman's class of beginners demonstrated the first steps in producing elementary sounds as gathered from tactile impressions of the finger tips on the bones of the face of the teacher and of the individual child during the phonation of *m*. Labial sounds, trills, differences in pitch, elementary vowels, etc., followed in a pedagogic order. The first principles in lip reading were also demonstrated with these young pupils. The children were all between four and six years of age.)

(Dr. Goldstein then showed and explained the audiometer chart and the graph of different types of deafness.)

An audiometer graph is made in the case of every pupil of the school, and one of the most useful features of the audiogram is to determine whether the child still retains a remnant of hearing within the speech range (300-3000 d. v.). It is emphasized, that if the audiogram indicates perceptibility within the speech range, such a pupil has a potentiality of stimulation and improvement when subjected to daily, intensified sound stimulation.

It is also of interest to note that the reactions to all of the cranial nerves are tested in each pupil with the hope that we may be able to differentiate whether a lesion of the auditory mechanism is central or peripheral. If it is found that all other cranial nerves (with the exception of the auditory) are functioning normally in a given case of profound deafness, it is almost fairly logical to assume by exclusion that the lesion is in the auditory field and not in the brain stem. If, on the other hand, in addition to a profound deafness you also have a defect in smell, in the motor muscles of the eye, or some impairment in the facial nerve, it is equally logical to con-

clude that there may be some lesion in the brain stem and not in the auditory field.

Each pupil is tested as to the activity of both the static and the acoustic labyrinth. Where we found that the static labyrinth, on tests by rotation or by douching, still shows a reaction and the presence of nystagmus, we are fairly safe in the conclusion that the acoustic labyrinth is not completely destroyed. If, on the other hand, there is a modicum of hearing indicating some reaction in the acoustic labyrinth, and if it is further found that the static labyrinth is inert, we assume that the modicum of hearing is still present, not because of the function power in the peripheral auditory mechanism, but because of some other source of sound conduction to the centrum that has not yet been determined.

To illustrate my point, I desire to demonstrate this girl of thirteen who has an absolutely inert static labyrinth in both ears, was born deaf, but in whom we have developed quite a substantial amount of hearing.

John Tyndall, the English master physicist, made the assertion that all of the organs of special sense were simply modifications of the sense of touch. We are substantiating this from many angles in our everyday observations in the schoolroom with the deaf child. He also stated that "light, heat, sound and electricity were all modifications of a similar wave of motion" and this, too, is being seriously considered in our pedagogy of the deaf child today.

Through the courtesy of the Western Electric Company, Scientific Equipment Division, there has been installed here a microphone and amplifiers by means of which we will demonstrate that even the breath sounds and the lowest spoken speech elements can be heard in the remote corners of this auditorium while the children with their immature voices speak through the microphone.

(Dr. Goldstein then gave a brief report of the functional tests, audiogram, clinical record and personal history of each pupil presented.)

(Miss Templemen demonstrates an older class of pupils. These children are doing sixth grade work, were asked question about the capital of Illinois, the population of Chicago, geographic position and other data comprehensible to the normal child doing sixth grade work.)

(Dr. Goldstein then demonstrated a boy of ten years who had been diagnosed as an aphasic, but on closer examination, after entrance to Central Institute, it was found that he had a marked defect in hearing. This pupil had been intensively trained, so that now he could hear fluent speech at about two feet from the ear. This was an illustration of some of the errors in diagnosis that the neurologist occasionally inflicts on the otologist and which can be corrected by careful pedagogy.)

In citing John Tyndall's theory of the correlation of the sense organs, I wish to emphasize the importance of sense training of every type. At Central Institute, we are trying to correlate the sight sense, the touch sense, the hearing sense, even when only remnants are present, and memory and the association of ideas that complement these special sense organs, psychologically and pedagogically.

We have incorporated all forms of sound stimulation into a wider and more comprehensive term and developed this larger field into a new system of teaching which we call "The Acoustic Method."

My definition of the Acoustic Method may be summarized in the following:

The Acoustic Method is a form of special pedagogy applied in the education of the deaf child in which every phase of sound is utilized for stimulation and education. The principal active factors applied in this pedagogy consist in the use of the human voice, various types of musical instruments (e.g., piano, organ, cello, bells, etc.) electrically activated tuning forks and, eventually, sound amplification as developed through telephone, phonograph and radio.

The human voice is the most selective form of musical instrument for sound stimulation in this pedagogy, but there are so many variations in quality and quantity of sound production in the use of the human voice, and its amplification often is so limited, that it cannot be used for constant daily stimulation without serious wear and tear on the part of the teacher.

We have developed at Central Institute for the Deaf a graduated system of exercises, and have set forth the details of the Acoustic Method pedagogically in a monograph which is at the disposal of all teachers of the deaf who undertake this special stimulation.

Since the advent of the radio and perfection of long distance telephony, the amplification of the human voice may be so perfectly controlled, that its application adds many new features as a stimulating agent in the conduct of this method.

In addition to voice stimulation, there should be prominently emphasized the importance of training the deaf child in accent, pitch and rhythm. The instrument of choice for this work is the piano.

(Miss Leila Raney demonstrated with a group of children around the piano the special steps developed in the production of pitch, accent and rhythm.)

I wish to emphasize the fact that we urge the beginning of training of the deaf child at as early an age as possible. His actual pedagogy may begin at three years of age. He has the same instinct and desire to express himself in some form of speech contact as the normal child. Psychologically, these instincts dominate in the deaf child at practically the same period as they do in the normal child.

The congenitally deaf child is frequently referred to as "deaf and dumb." Dumbness is not a pathologic entity and is only a consequence of the child's deafness and his inability to imitate the sounds of the human voice. Every congenitally deaf child has the potentiality of speech.

Speech in the deaf child, unless a sufficient remnant of hearing is present, must be mechanically produced. We are now in a position to demonstrate the fact that this form of mechanical speech in a child with profound deafness may be so perfectly produced, that pitch, volume, inflection, fluency and quality are brought forth so that the speech of the deaf child becomes intelligible, pleasant and almost normal even to the untrained ear.

Another observation to be emphasized is the fact that if the congenitally deaf child is not taught by speech methods until after he is eight or nine years of age, there may already be present a beginning ankylosis of the speech mechanism and a beginning atrophy of the intrinsic muscles of the larynx. When such pathologic handicaps exist, the opportunity for development of normal speech production in the deaf child is materially lessened. We urge, therefore, that the deaf child be sent for special training as early as possible. At Central Institute, we admit them for instruction as early as three years of age.

Referring again to the Acoustic Method, it is interesting to recall that the evolution of this special form of sound stimulation must be credited to the otologic profession and not to the pedagogues of the deaf. The eminent French otologist, Itard, was the first to suggest sound stimulation where partial deafness existed in the pupil. In 1805, Itard began his acoustic practice with bells, the rhythmic beat of the drum, the sustained notes of the flute and, finally, elementary sustained vowels of the voice. In 1832, Blanchet and Deleau declared themselves in favor of such acoustic exercises in the education of the deaf. Beck, Jager and Wolff interested themselves in this special phase of training in Germany. In England, Toynbee and Wilde took up this question.

The most important accomplishment in this field in its evolutionary period must be credited to Urbantschitsch of Vienna. In 1893, Urbantschitsch demonstrated before the Vienna Medical Society a group of deaf children trained under his supervision by the teachers of the Döbling Institute for the Deaf. I recall the interest of the assembly on this occasion, the skepticism of several distinguished authorities, the challenge of the status of total deafness of the pupils demonstrated, the refutation by Urbantschitsch in his determination of complete deafness preceding the period of practice, the excellent response of the pupils themselves, and the general impression and conclusions by doctors and teachers present of the value of this work.

Today, the Acoustic Method, as the result of these various evolutionary changes, its critical analysis, its systematic production, the employment of sense training, the inclusion of pitch, accent and rhythm work with the piano and stimulation by phonograph, telephone and radio, has become a surprising and formidable factor in the education of the deaf child. We are still hovering over the threshold in the development of this special training, and I predict splendid accomplishments in its further development.

Even if we deal with a remnant of hearing in a deaf child and accomplish the limited step of securing a differentiation of pitch of voice, we pass an important milestone in the speech training of that particular pupil, for this pitch differentiation alone will greatly assist the teacher in overcoming in that

pupil the peculiar monotone and wooden character that has been so much in evidence in the speech of the deaf.

I point with justifiable pride to the teaching staff which we have developed at Central Institute for the Deaf. Our teachers are the product of our own Normal Training School and our entire staff of twenty-one teachers have been carefully trained, not only in the principles and practice of special pedagogy for the deaf, but they are conversant with the anatomy and physiology of the speech mechanism and the hearing organs, and most of them are as familiar with the organ of Corti under the microscope as are our postgraduates in medicine, including many otologists.

I say deliberately: There are but two thoroughly organized, systematically conducted Normal Training Schools for oral teachers of the deaf in America holding to a curriculum which is thoroughly adequate. One is the Clarke School at Northampton, the other is Central Institute for the Deaf in St. Louis, and I heartily endorse the statement of Dr. Caroline Yale, the eminent principal of Northampton: "If you want to produce better speech in deaf children, train your teachers with better qualifications for this important work."

In our regular and postgraduate normal training classes during the past ten years, Central Institute for the Deaf has taught over four hundred teachers, offering them such modern pedagogic data as we have been able to gather in our observations of the deaf child. We consider our training school one of the most important machines for the advancement of the education of the deaf, for these teachers radiate to every school from Maine to Florida and from coast to coast, and our work has even been carried back by able exponents to Mexico, Canada and England.

I have emphasized the importance of sense training as a part of the education of every deaf child, and I wish to demonstrate to what degree sense training can be made practically applicable. To educate tactile impressions to a high point of efficiency is simply to practically corroborate the thought of John Tyndall that "all organs of special sense are but modifications of the sense of touch." Professor Gault of Northwestern University, in Chicago, demonstrated on normal university students that the touch sense could be educated to receive impressions of spoken words, and in the course of some months a group of such students were able to differentiate

some twenty odd words spoken through a long distance speaking tube and received by them as tactile impressions on the hand and fingers incased in a sound proof box.

We will demonstrate a more advanced accomplishment in a young deaf girl of nine years of age. Marie is totally deaf and her audiometer record is here for your inspection. For these experiments I have used a large simple megaphone such as used by cheer leaders and baseball announcers. Over the larger end of the megaphone a disk of heavy Whatman paper is tightly stretched. The teacher speaks through the mouth-piece of the megaphone. The pupil touches the drumhead of the megaphone lightly with the finger tips. The pupil's eyes are blindfolded so that she cannot read the teacher's lips. The experiment begins with elementary voice sounds and continues through various states until we reach the production of complete sentences. In this experiment we develop every pedagogic accessory including sight, memory, speech and association of ideas.

We now offer the megaphone experiment in tactile impressions of speech with little Marie Tilson. (The demonstration was begun by a differentiation of elementary vowel sounds ā, ō, ä, ē, o͝o; differentiation of pitch of vowel elements; reproduction of thrill, guttural, labial and other sound combinations; individual words, such as *mother, paper, railroad, teacher;* geographic names, *New York, St. Louis, Chicago, Philadelphia;* proper names, *Dr. Goldstein, Miss Arnold, Miss Connery, Dr. Norval Pierce*—a name just given to the child and differentiated in conjunction with other names of this group and given in unexpected series).

Finally, a group of fruit charts were exhibited on a rack. The teacher spoke into the megaphone: "Show me some apples." and the pupil promptly pointed to the chart with apples and pronounced the sentence. "Give the bananas to Dr. Goldstein." The act was carried out and the sentence spoken. "Place the oranges on the table." "Give the grapes and the bananas to Dr. Norval Pierce.")

In conclusion, I think you will agree with me that some of the demonstrations and experiments which we have presented to you tonight offer an unlimited field for research and much food for reflection. The future of the speech trained deaf child is no longer in the balance and from now on there should be the most encouraging and helpful evolution in his pedagogy.

DISCUSSION SYMPOSIUM ON DEAFNESS.

DR. J. A. STUCKY, Lexington, Ky.: It has not been so much due to lack of desire on the part of the medical profession that more progress has not been made, as to a lack of enthusiasm and determination to solve the problem which for ages was considered both hopeless and impossible. More progress, helpfulness and happiness for the socalled deaf-mute has been made in the past ten years than in all the previous history of otology. In making this statement I speak chiefly from the results in my own state, upon the otologists, citizens of Kentucky and inmates of our State School for the Deaf.

Great intestest was aroused a year ago by the addresses and demonstrations of Dr. Goldstein in Louisville, Lexington and the School for the Deaf at Danville, Ky. The examination of several hundred children at this place, by myself and associates, revealed a large percentage of cases with pathologic and functional conditions which could be removed or improved.

We know many cases of deafness due to middle ear disease can be arrested, and often improved after the disease has been arrested, and "I have seen many cases improve after intestinal disturbances were cleared up." Besides this, we often find, as a decided etiologic factor, focal infection as well as an endocrine imbalance resulting from nutritional deficiency. I emphasized this point in my discussion of Dr. Drury's paper, in the Section of Oto-Laryngology at Atlantic City, May, 1925. The majority of these cases have no clinical evidence of a pathologic condition, and as has been pointed out, none has been demonstrated by autopsy. We are finding increasing evidence that metabolic changes, in which the endocrines are involved, have much to do with this dysfunction. This has not as yet been demonstrable in the laboratory, but clinically it has been shown to be true. Besides the cases mentioned by me in Dr. Drury's paper in the Section of Oto-Laryngology at its last meeting, I have been able to check up several others which show decided and increasing improvement as a result of a well balanced diet with endocrine feeding.

There is no doubt that the psychologic state of the patient has a great deal to do with the ability to hear, and the proper handling of such a patient from the psychologic standpoint has meant improved hearing. Nor is there any doubt that many intranasal operations, X-ray treatments, massage, etc., have seldom resulted in more than temporary improvement.

The most hopeful sign of the times is the awakening of the medical profession and the people, and I agree with Dr. Rypins' statement that "a cure means education for the deaf people themselves," and until this is done and the deaf refuse treatment at the hands of these pseudo-scientists, little constructive work can be accomplished. Numerous people need not only lip reading and financial aid, but must be mentally rehabilitated, and when our hearing tests are standardized, so that all of us will take them in the same way, and educators and the medical profession work side by side in the prevention of deafness and the welfare of the deaf, we will have made a decided step forward in otology.

I am heartily in accord with all that Doctor Goldstein has said, and the demonstrations made prove conclusively that the problem of the deaf

is being solved, and there is now more than hope for the large number of humanity who are living in a dead world because of inability to hear. The solution of the problems of the deaf will be hastened in proportion as otologists are interested, the people educated through civic clubs, especially the Women's Clubs, and our legislative bodies made to give such financial aid as will enable us to convert the class thus handicapped into assets instead of liabilities.

I feel that as medical men we do not appreciate as much as we should the importance of idealism in this work, and until we do, we cannot impart to the public the necessity of its doing its part as it should.

Dr. Norval H. Pierce, Chicago: I have nothing but the most profound admiration for the work that is being done by Dr. Goldstein. I have witnessed his development, and at one time I thought he was more or less of a fanatic rather than a healthy enthusiast, but more and more I see now that he has at length achieved what was regarded a few years ago as the impossible.

As to whether the small residue of hearing that he may discover or imagine in these cases has very much to do with hearing, I am somewhat doubtful. I think that Tyndall's idea regarding the organs of hearing and other specialized senses—that they are merely modifications of sense of feeling—is the correct one. How much this mysterious tactile sense can be educated and substituted for hearing, we have no idea at present. Perhaps there is a certain amount of telepathy that goes with this matter of the deaf child hearing. It perhaps merges into psychology, and from that standpoint advance may be expected.

From that attenuated atmosphere of thought, we come down to what we can do for the hard of hearing—what we otologists can do for the very hard of hearing in the way of treatment. It is not a rare disease by any means. It is a very common disease and in my opinion it will, as hereditary diseases are likely to do, increase as the population increases in numbers. We know nothing whatever about the cause or the nature of otosclerosis, and therefore we can do little or nothing for it in the way of treatment.

I believe it is a good time for me to announce, Mr. President, that I am the Chairman of a commitee appointed by the American Otological Society, to begin a rather extensive investigation of otosclerosis. Dr. Arthur B. Duel of New York, also a member of that commitee, approached the Carnegie Foundation with a view to obtaining a grant to this committee. About a week ago, I received word that this committee had been granted the income of $500,000 for five years, to be devoted to research in, and investigation of otosclerosis. A part of this money is at the disposal of any man belonging to this organization, provided his problem is deemed sound and constructive.

We are going to make a wide appeal to the otologists and pathologists of this country, in an attempt to secure material for pathologic study. We are going to appeal to the Federation for the Hard of Hearing, to see if we cannot get otosclerotics to bequeath their ears to us. And they should do this in an endeavor to check this disease, from which their children will probably suffer. We are going to make a biologic analysis of these people, and endocrine investigation. We are going to study more fully and care-

fully and minutely the temporal bone, in which this disease has its situation. We are going to solve some of the problems that are at present unsolved regarding the circulation of the capsule of the labyrinth. We are going to study something that no one knows anything about, and that is the relationship of fluorin to the normal and pathologic processes of bone. We will study the calcium content of the blood, its secretion and excretion. We will endeavor to make a more accurate chemical analysis of bone than has heretofore been available. We will endeavor to discover the manner in which these elements are tendons or bony tissue. All this is fundamental to the study of otosclerosis.

This is the direction in which we must go if we hope to benefit the otosclerotic therapeutically, not by sitting under the lamp in our pleasant libraries and getting up a lot of pretty speeches—we must get down and dig and sacrifice, without hoping for reward other than the satisfaction of knowing we have helped, or at least attempted to help, in advancing the knowledge of disease. We can do very litle. Dr. Harold Hays quoted me as saying that there was not one case reported in this country of otosclerosis that had been proved at autopsy. It was not said in support of the belief that otosclerosis is a rare disease. It was said in derision of the medical profession—that there had not been one case of otosclerosis posted in this country in which the temporal bone has been studied microscopically. That is due to two reasons: First, it is very difficult to get autopsy material because otosclerosis is not a fatal disease —death occurs from some intercurrent disease. Second, the absolute lack of spirit on the part of the otolaryngologists as a whole in real investigation work, research work that involves material sacrifice And unless we are willing to sacrifice some by going into the laboratory and doing something that we know we will not get money for, we are not going to advance as we should. The spirit today among medical men—otologists and rhinologists as much as those in other branches—is too often the spirit that animates the stockbroker, and that is at variance with the professional spirit. What are we going to do for otosclerosis today?

Dr. Harvey Fletcher, New York City: It seems to me we have had a rather wonderful day for the hard of hearing.

It may be true, as Dr. Pierce said, that little can be done for the hard of hearing by medical treatment. One of the principal things we can do as organized bodies, is to stimulate interest and point out what needs to be done, for such work will bring forth grants from various foundations such as has been described here tonight by Dr. Pierce. It is significant, that during the last two or three years since a number of people have been driving hard at this thing, these grants have been made to medical societies and other institutions interested in the welfare of the deafened, and they are not yet at an end. So it seems to me, that the education of organized bodies to the needs of the hard of hearing is to the point and has effect now, both in creating funds and also in pointing out means for future work in this line.

With reference to the school child, there are three parts to the problem. First we must develop some method of finding the child who needs attention. In my judgment this development is a proper problem for the physicists and engineers. In the past, trained physicians have been

trying to solve it, but it is not the kind of work that they are educated to do properly; it is a little out of their line.

When we find the child, what are we going to do with him? The second part of the problem is to determine what medical attention should be given such a child and to determine how this can best to accomplished. This is, of course, the business of the physician. Then, something must be done for his education, and that is neither a problem for the physicist nor the physician; it is a problem for the educator. These three, namely, the physicist, the physician, and the educator must work together co-operatively to solve the problem satisfactorily. A significant sign of progress is the fact that we now see the cooperation between these various branches of science that has not existed before.

Things are moving much faster than when I first became acquainted with this problem, three or four years ago, and if all those who are interested will keep talking and stirring up enthusiasm, I am sure we will obtain the solution of the problem.

ORATION.

THE PATHOLOGY OF DEAF-MUTISM.*

By Professor F. R. Nager, M.D.

UNIVERSITY OF ZURICH, SWITZERLAND.

Before entering upon the subject of my lecture, I wish to express my cordial thanks for the honor of your invitation to take part in your annual meeting as your guest. I am fully conscious of the distinction conferred, but also of the duties connected with my task, and trust that I shall fulfill your expectations.

I should like, however, to bestow the greater part of this honor upon my two teachers, the deceased Professor Bezold of Munich—whose remarkable textbook has been translated excellently in Chicago by our colleague Dr. Hollinger—and upon Professor Siebenmann in Bâle, whom many of you will remember personally from his visit to Boston in 1913. With heartfelt gratitude I would recall to your memory the great merits of this prominent Swiss otologist.

My scientific pedigree prompted me to choose the subject of deaf-mutism for this lecture, because through their research work Bezold greatly advanced the clinical, and Siebenmann the pathologic aspect of this subject.

The pathology of deaf-mutism, about which I should like to give you a general survery, is a new field of work in otohistology, which could be entered upon successfully only after the introduction of the celloidin technic. The macroscopic changes in acquired deaf-mutism were well known to the older investigators, but the minute alterations in the labyrinth of congenital deaf-mutism could be ascertained only by the modern microscopic technic.

In the first decade of our century, when this research work became known, it was supposed that certain alterations were characteristic of deaf-mutism, just because similar changes

*Illustrated with lantern slides.

were found in the labyrinth of congenitally deaf animals, for example Japanese dancing mice, albino cats and dogs, etc. Later on, however, it was shown that analogous abnormalities may exist also in cases of simple deafness. Furthermore, similar anomalies were described in experimental labyrinth pathology, and to this discovery we owe much insight into the pathogenesis of inner ear diseases.

It may be added, that in isolated cases histologic research did not result in finding any local changes. It is supposed, therefore, that there must exist anomalies of the central connections and brain. According to present requirements, examination of the inner ear should be followed by an examination of the central connections. And, indeed, the neurologists repeatedly ascertained changes in these structures, though it remains for future examinations to demonstrate which alterations are primary or secondary, and which are the cause of the defect in hearing.

The "Pathology of Deaf-Mutism" for this reason does not represent something that is entirely confined to the labyrinth of the deaf-mute. A résumé under this designation offers nevertheless so much that is instructive and interesting, especially when experiences gained in the other researches of otohistology are also utilized, that even future otologists will continue to cultivate this interesting field.

In the same way, the conception of deaf-mutism, though accepted generally as a clinical term, scientifically is not quite correct, because according to Bezold's hearing tests, only a small number of socalled deaf-mutes are in fact totally deaf; the latter belong mostly to the acquired forms of deaf-mutism. Quite a large number of the socalled congenital cases, on the other hand, exhibit very considerable remains of hearing, available for aural education.

There exists, therefore, no distinct line of demarcation between deaf-mutism and simple deafness, not only in their pathologic but also in their clinical aspect. It is not the high degree of deafness alone, but its existence in early childhood, when speech should be acquired with the aid of hearing, that leads to deaf-mutism.

I should like to disregard in this lecture the central disturbances and their clinical effects on the faculty of hearing and the acquirement of speech, and to limit myself entirely to deaf-mutism and its local manifestations in the ear. But I

should like to emphasize, nevertheless, that future deaf-mute examinations will have to include still more thoroughly the central disturbances and their histologic changes.

For a long time, a distinction was made clinically between congenital and acquired deaf-mutism, which proved to be very useful, but which is not scientifically exact on closer scrutiny. We depend here predominantly on the statements of the child's parents, which are often unreliable. In addition, a certain number of parents very unwillingly admit, even if they know it, the congenital nature of a defect. Moreover, it is not always possible to decide, even by clinical examinations, whether deaf-mutism be congenital or acquired; there will always remain uncertain cases.

The hope of finding criteria in the microscopic investigations, which would permit of definite distinction between these two forms, also proved to be deceptive, because similar histologic changes could be found in both forms. With increasing knowledge of the histologic abnormalities, the need arose to group them in some manner. This, however, met with considerable difficulties, especially as to the classification of congenital deaf-mutism. According to our knowledge today, there is as yet no method of classification, which satisfies all demands, and at the same time embodies a proper consideration for clinical, etiologic, histologic and pathogenic points of view.

We will, therefore, have to group the cases, as we become interested in special questions, in such a way that we may understand how analogous pathologic changes may be found under quite different etiologic factors. I shall now submit to you a certain classification in accordance with the work of Siebenmann, taking into consideration the results of late researches.

Acquired deaf-mutism is today explored most thoroughly, because the results of the general and experimental labyrinth histology have substantially assisted in clearing up this field. We deal in most of these cases with the results of inflammatory processes in the labyrinth. Their origin may be meningitic, tympanic or hematogenic (metastatic). Noninflammatory affections, as for instance injuries of the skull, are the cause of labyrinth disease in the minority of cases.

As far as labyrinthitis is concerned, we know today all its grades and different stages up to a state of healing, so that

we are able to interpret the alterations found in deaf-mutism arising from this cause. The meningitic cases are most frequent, because a number of diseases to be considered in this connection, may occur as epidemics, (meningitis cerebrospinalis, scarlet fever, measles, parotitis, typhoid).

Deaf-mutism caused by meningitis has been known longest and best. Each epidemic was always followed by an increase of deafness, or of deaf-mutism. In such cases we find a new formation of bone and connective tissue of different degrees up to the complete obliteration of the labyrinth cavities. If the cochlear duct is preserved, it will show important changes, such as collapses, dilatations or adhesions in its interior; Corti's organ is missing, so are the cells of the stria vascularis. The membrana tectoria is often enclosed in an epithelial capsule. The spiral ganglion and nerve fibers are absent. In isolated cases, a fistula into the middle ear could also be traced.

That scarlet fever and measles can likewise lead to meningitic labyrinthitis had to be assumed from the results of clinical examinations of deaf-mutes with normal drumheads. I shall later on demonstrate some slides of a case of deaf-mutism following scarlet fever meningitis, showing almost complete bony obliteration of the labyrinth. In cases of measles, bronchopneumonic complications are especially to be feared. and these may be followed by metapneumonic meningitis with consecutive deaf-mutism. Moreover, deaf-mutism after parotitis and hereditary syphilis seems to be due to meningitic labyrinthitis, according to the clinical and the few microscopic investigations.

Tympanic labyrinthitis also occurs frequently, and has become well known as a cause of acquired deaf-mutism. Every necrotic otitis media endangers the inner ear through the possibility of an invasion of the labyrinth. All of us are familiar with such cases of deafness in childhood, followed by deaf-mutism, after scarlet fever, measles, influenza otitis, as well as after tubercular and congenital syphilitic middle ear suppuration.

Histologic examinations disclose very extensive destructions even up to an obliteration of the labyrinth, not infrequently coupled with cholesteatoma. Aside from the new formation of bone and connective tissue, the route of labyrinthine infection may be found as a healed fistula. Not infrequently the remains of the membranous labyrinth can be

traced, the changes being similar to those found in cases of meningitic labyrinthitis. A metastatic labyrinthitis has to be accepted as the cause of deaf-mutism after osteomyelitis, but, as yet, this view has not been confirmed by histologic investigations.

In cases of noninflammatory labyrinthic affections, injuries to the skull in earliest childhood assume first place as a cause of deaf-mutism. Here, also, labyrinth pathology has greatly clarified our knowledge. A manifestly greater importance has of late been attached to injuries during birth, since the labyrinths of children who have died intra- and postpartum may show changes which correspond entirely to those found after severe injuries of the skull.

The labyrinths of traumatic deaf-mutism are partly filled with bony and connective tissue; the scala media in a case observed by us showed tremendous dilatation; the spiral ganglion and nerve fibers had been destroyed almost entirely. The conditions correspond to the after effects of inflammatory labyrinthitis.

From the clinical investigations of deaf-mutes and from the histologic examination of persons who became deaf, we have to reckon also with the possibility that isolated affections of the auditory nerve, as well as of Corti's organ, may lead to acquired deaf-mutism, as the result of a neuritis with consecutive atrophic degeneration. Such postinfectious or toxic neuritis not infrequently appears after the disease previously referred to (meningitis, scarlet fever, measles, diphtheria, parotitis, typhoid). Further, a toxic neuritis of the cochlear nerve would explain the presence of a normal drumhead with normal reaction of the static labyrinth in these deaf-mutes. Exact histologic investigations of such well defined cases have not as yet been described, but findings of that character may possibly have been included in reports on certain cases of congenital deaf-mutism.

The pathology of congenital deaf-mutism is much more many sided. First of all, I would like to comment upon endemic congenital deaf-mutism, since the clinical, pathologic and etiologic aspect is well known in Switzerland. Here we are dealing with one of the symptoms of endemic cretinic degeneration, not uncommon in certain mountainous regions (i.e. in the Alps of Central Europe, and in Central Asia) hav-

ing similar hygienic, economic, geologic and climatic conditions.

The central nervous system, the thyroid gland, the skeleton and the ear being affected in different degrees, afford us a varied picture of endemic cretinism, which, in its greatest development, is represented by the idiotic, goiterous, deaf-mute dwarf.

The pathologic changes in endemic congenital deaf-mutism affect in a very peculiar way the medial tympanic wall. There is a marked hyperostosis of the promontory, with a narrowing of the window niches with adhesions of the deformed stapes. In the cochlea, a small hyalin body, which is embedded between Corti's organ and the membrana tectoria, has been hitherto found only in endemic deaf-mutes. Except for this, the spiral organ of Corti and the nerve fibers may be normal, as such patients generally have considerable remains of hearing.

These striking changes in the ear are probably due to noxious influences in human development, during the last third of the fetal period. Neurologists also have reached similar conclusions, based on the results of histologic investigations of cretinic brains.

These changes are so typical, that the diagnosis of the endemic form becomes possible also postmortem and without clinical data. In cretinic dogs, similar abnormalities have also been ascertained. It may been mentioned incidentally, that in Switzerland the most severe forms of the endemic cretinic degeneration are steadily decreasing, due to persistent improvements of hygienic conditions, the opening up of isolated districts, and the fight against endemic goiter with prophylactic iodin therapy.

In sporadic deaf-mutism the changes are exclusively in the labyrinth, mostly in both ears. They have to be looked upon as malformations, though it is not certain as yet to what extent they are to be considered as congenital defects or as consequences of intrauterine inflammations. The alterations may exhibit different grades of development, which have led to a division into different groups and types.

The complete absence of the labyrinth and pyramid is very rare (Group A). In the more frequent malformations of the auricle (microtia and atresia auris congenita) the inner ear is less affected or not at all.

Much more numerous, on the other hand, are the slighter changes in the labyrinth, which are either confined to the neuroepithelium of the scala media of the cochlea (Group B), or may be associated with anomalies of the sacculus as well as of the cochlear duct. (Group C). The opinions about the congenital character of the changes in Group B have begun to waver lately. Early postfetal degenerative atrophic processes in the eighth nerve and its neuroepithelium, perhaps also birth injuries, might play here a causal rôle.

The third group (C) with sacculocochlear degenerations contains the greater part (70%) of sporadic congenital deaf-mutes with remnants of hearing. It includes most of the constitutional hereditary form. Here also different grades of development are observed: those with intact modiolus, those with defective central axis, and finally those with large dilations of the saccus endolymphaticus (Type Mondini). The last can easily be observed macroscopically, and has attracted the attention of older authors. Here, even the bony labyrinth is much affected. The neuroepithelium, the spiral organ and the nerve fibers likewise show extensive degenerations. In the cochlear duct, there are folds and adhesions between Corti's organ and the stria vascularis. The utricule and the semicircular canals are normal; so are their functions.

Certain similarities to the changes in cases of meningitic and experimental labyrinthitis caused Siebenmann to assume intrauterine inflammatory conditions in the labyrinth or in the cranial fossae as the cause of a number of these alterations.

Besides these forms, very marked malformations of the cochlea may exist, as for instance the presence of only one and one-half coils, instead of the normal two and one-half coils.

Quite analogous results were obtained from the histologic examination of congenitally hard-of-hearing patients. It is very interesting to observe that tuning fork examination revealed defects even in the interior tone limit in cases in which the upper coils of the cochlea were absent. It may be added that in quite a considerable number of these cases the bony labyrinth contains distinct spots of otosclerosis. This is probably not due to accident, as we know that on the other hand the cochlea of otosclerotics very often shows slight malformations.

It must be left to future investigations with the aid of more observations and a faultless technic to shed an increased light, especially on the etiology and pathology of all these forms.

To effect this, we shall have to take into consideration the results of the general labyrinth pathology, secondly of other ear and brain malformations, and lastly of experimental and comparative anatomic studies in general.

I have attempted to outline a survey of the pathology of deaf-mutism. It is still a mosaic, like ancient pictures, showing many a gap, rendering a general impression very difficult. But if we all, not only we in the Old, but also you in the New World, with its magnificently equipped laboratories, will continue to work ahead, then a more perfect picture in all its details will be unveiled.

This knowledge surely is not only of scientific but also of practical interest, because from it therapeutic and prophylactic viewpoints will arise, which probably will reduce the frequency of deaf-mutism.

I wish to express the hope, that this short lecture has awakened in you the interest and the desire for mutual work, through which the high aim which you seek to effect by an international exchange of thoughts and experiences may be materially advanced.

FORMS OF OPTIC NERVE ATROPHY.

EDWARD JACKSON, M.D.

DENVER, COLORADO.

When this subject was suggested as a good one to be brought before the Academy, the writer thought of optic nerve atrophy as a group of conditions distinct from other pathologic states; which was capable of subdivision in a way that might help us to bear in mind the main features of the different kinds of optic nerve atrophy, and in their recognition clinically. The survey of the subject, made with this in view, has changed his ideas about this; and a review of some facts revealed by that survey may have value for others.

Wilbrand and Saenger devoted volume five of Die Neurologie des Auges to "Die Erkrankung des Opticusstames," literally the falling sick of the optic stalk or stem. In this volume, their classification begins with inflammatory affections, acute, chronic and hereditary; perineuritis, with interstitial peripheric neuritis, independent or descending neuritis and complete transverse optic neuritis. They discuss the etiology of simple optic neuritis, and then take up the atrophic conditions of the optic stalk and progressive atrophy of the optic nerve. Rupture or division of the optic nerve, they have considered in volume three under the general diagnosis and symptomatology of amblyopia and amaurosis.

Parsons' Pathology of the Eye discusses the degenerations of the optic nerve under headings, Senile Degeneration; Secondary Atrophy; Primary Atrophy of the Papilla and of the Nerve Trunk; and Vascular Degenerations. Fuchs, in his textbook, recognizes two main varieties of optic nerve atrophy —simple and inflammatory. De Schweinitz' Diseases of the Eye mentions as varieties of optic nerve atrophy: Primary Atrophy (sometimes called gray, progressive, spinal or tabetic atrophy); Secondary Atrophy; and Consecutive Atrophy, including (a) Postpapillitic Atrophy and (b) Retinitic and Choroiditic Atrophy.

Weeks' Diseases of the Eye thus discusses forms of optic

nerve atrophy. "Atrophy may be preceded by inflammation of, the nerve manifest at the intraocular end of the nerve, papillitis, consecutive or postneuritic atrophy; or it may come on without visible inflammatory disturbance consequent on disease of the nerve back of the globe, of the chiasm, tracts, or basal ganglia, simple atrophy, descending atrophy. It may also follow certain diseases of the choroid and retina, secondary or ascending atrophy."

"Although visible inflammatory changes in the ocular end of the optic nerve are seldom seen in "simple" optic nerve atrophy, it is undoubtedly a fact that slight congestion is present in the earliest stages in many cases, and in all probability a slow inflammatory condition exists in some part of the nerve. This is particularly true of hereditary optic nerve atrophy, the atrophy of multiple sclerosis, and of general paralysis of the insane (Gowers). Simple atrophy occurs more frequently in males than in females, and more frequently in adults than in children.

Simple atrophy may be classified as primary in those cases in which the loss of vision keeps pace with the changes in the disc, and secondary in those cases in which the "structural integrity of the nerve" is interfered with by trauma or some other cause, the atrophy being visible some time after vision has partly or wholly failed. The two forms cannot be differentiated by the ultimate appearance of the disc. The secondary atrophies are much more frequently unilateral."

Atrophy is an organic degenerative change, marked by shrinking with loss of characteristic structure and function. It is the end and result of many morbid processes. Like death, it belongs to many diseases that may have little else in common. Its classification is impossible in the sense that living organisms, complete diseases and many natural phenomena are classified; and the causes for diversity and confusion, in attempts to classify atrophies of the optic nerve, become apparent when we consider the different bases that have been taken for the classification of diseases.

Anatomic Classification. The grouping of diseases according to the part affected is recognized in the term atrophy of the optic nerve. Different subdivisions of the nerve may be made, as intrabulbar, intraorbital and intracranial portions. Or we may divide the nerve into axial and peripheric zones, as is often done in discussing neuritis. Or we might divide

processes according to the tissues entering into the optic nerve; like the old classification of parenchymatous and interstitial inflammations, which is reduced to absurdity in regard to the cornea, where the same inflammation is termed indifferently by different authors, parenchymatous or interstitial. In the optic nerve we have within the neural sheaths the nerve fibers, with axis cylinders and medullary sheaths, the glial supporting tissue intimately associated with the nerve fibers, the connective tissue septa dividing the nerve into its chief bundles and carrying the chief blood vessels, and the vessels themselves with their various coats and contents.

It is conceivable that each histologic constituent of the optic nerve might be the starting point, or the seat of the characteristic changes of some form of optic atrophy. But we know so little of the order in which these different histologic elements are affected, and of the significance of changes found in each; we are so little able to discriminate among them as to cause and effect, that they furnish little basis for a system of classification. The subject is yet further complicated by the acceptance of the neurone as the anatomic and still more the physiologic unit of the nervous system. The nerve fibers of the optic nerves are portions of axones belonging to the ganglion cells of the retina, and to a very limited extent, of the primary optic centers. The general theory of axones is that they depend for development and nutrition upon the cell body situated quite outside the optic nerve.

Pathologic Processes offer but little better basis for classification. Their earlier stages are unknown. Only when disease has passed from its peculiar functional manifestations to its more uniform, general and least characteristic organic changes, do the departures from normal become recognizable by our methods of studying pathology. About all that has been done in this direction has been to recognize an order of succession in pathologic changes, on which have been based the distinction between primary, secondary and consecutive atrophies.

But atrophy, like death, is always a result of antecedent morbid processes. In a broad sense it never can be primary. Whether it follows neuritis, papilledema, mechanical pressure, poisoning or other noxious change in the nutritive fluids, or an essential pathologic change in the nutrition of the nerve cell, it is always secondary. All that can be said of primary

atrophy is that its antecedent pathology is unknown. Secondary atrophy on the other hand is not the first discoverable pathologic change in the case. All atrophies of the optic nerve are secondary to pathologic changes in some part of the body. It seems only allowable to call "primary" those atrophies not preceded by noticeable pathologic changes in the optic nerve itself.

In considering a classification of optic atrophies by pathologic changes, it must be remembered that the optic nerve is not a peripheral nerve at all, but a part of the central nervous system. In common with the brain, its immediate supporting tissue is neuroglia; and the glial cells are not stimulated to proliferation by irritants, as are the fibroblasts of connective tissue. Hence we have failure of neuroglia to compensate for shrinking of nerve fibers, with the formation of glial cysts in the brain, and cavernous spaces in the atrophic optic nerve. It is conceivable that cavity formation might be a form of atrophy beginning in the glial tissue, but the weight of evidence indicates that the nerve cell elements, the axones, are the tissue primarily affected; and that their shrinking merely is not made good by any glial proliferation.

The proliferation of fibroblasts, increasing the thickness of the connective tissue septa of the optic nerve, may be a primary step in some optic atrophies; and justifies the consideration of peripheric neuritis apart from axial neuritis, the former involving the connective tissue near the nerve sheaths, and the latter arising in the nerve fibers under the influence of injuries by chemical poisons.

Clinical Classifications have been attempted; "white" and "gray" atrophies, "postneuritic," "postpapillitic," "retinitic," "choroiditic," "progressive," "atrophy with excavation," etc., are instances of attempts in this direction. Such classifications must vary with the particular clinical feature on which they are based, and have little significance in any other relation. Such designations are often convenient to use, to call attention to some outstanding feature of a particular case. But if they are taken as each indicating a class of cases, more or less distinct from other optic nerve atrophies, they can only lead to confusion, and hinder the discovery of differences in anatomic susceptibilities, or deeper pathologic relationships. They are likely to conceal the etiologic relation-

ships of optic nerve atrophies which are of the greatest importance, both for theoretic and practical consideration.

Etiologic Classification. The frontier of modern medicine, over which its great advances have been made, is that of etiology. From our growing knowledge of the causation of disease begins to emerge a practical system of classification. Etiology links up closely with tissue liability, hereditary or acquired; with tissue reactions and pathologic changes; with symptomatology; with the progress of the case, its diagnosis and prognosis; and above all with its treatment, the eminently practical object of medical art. This applies perhaps more to the terminal process of disease, atrophy or death, than to the classification of complete diseases. It is too soon to erect a complete system for the classification of optic nerve atrophies, but the etiology may be stressed in all our studies of optic nerve degenerations and atrophies.

By fixing the cause of each case of optic atrophy reported, either with certainty or probability, we can prepare it for classification in its proper group, as the important relationships of etiology are recognized. Abiotrophy, trauma, pressure, toxic agents, infections (acute and chronic), senile and degenerative changes, general nutritive influences, endocrin imbalances, are all suggestions of a classification that will be at once thoroughly scientific and highly practical. At first, we may find it only possible to say "atrophy from measles" or "atrophy following mumps." But in time we will be able to place the cases that belong together, in one group, to work out the relations of groups, to recognize resemblances of pathologic processes, community of symptoms, similarities of course; on which valuable associations of atrophies having different origins can be based. To keep in mind the cause of a pathologic process opens the mind to observation of its course and resemblances, and impresses the significance of possible differences in its exact mechanism.

Summary. Atrophy of the optic nerve is the terminal stage, so far as the optic nerve is concerned, of many distinct disease processes. Therefore, it does not need or lend itself to formal classification, as do living organisms, or specific diseases. We do not know enough about the histopathology of the different tissues making up the optic nerve to base a final classification on the changes in each of these tissues.

Atrophy of the optic nerve is the goal, to which converge pathologic processes of wide variety, of causation and early course. The most valuable classification we can make for it, is of the causes that may set up the processes which tend toward this goal—the etiologic classification. This may be admittedly complex and incomplete, but it is the most widely serviceable; and as our knowledge of the different causes of atrophy becomes more complete, this classification is capable of growth, refinement and unification.

Meanwhile, it is right to use such terms as primary, secondary, simple or inflammatory, in designating different forms of atrophy only with the clear understanding that these are adjectives relating to certain aspects of the case under discussion, and not the divisions of a rigidly scientific system of classification.

DISCUSSION.

Dr. William Zentmayer, Philadelphia: Dr. Jackson has shown the confusion in the terminology of optic atrophy existing in the text books he has quoted. Had he quoted further, it would have been confusion worse confounded.

Dr. Jackson's paper is timely and thoughtful, but disappointing in not suggesting a tentative solution of the problem as a working basis until the time has come (which I fear is in the very remote future) when, as he says, the terminology may be based upon the sound foundation of etiology.

One teaches his students what he considers a fairly logical classification, but later the student finds in his reading that what he was taught to consider, for instance, a secondary atrophy, is applied by someone else to another form of atrophy, as shown in Dr. Jackson's quotations from de Schweinitz and Weeks.

Some of this confusion comes from the employment of the terms "secondary" and "consecutive." I try to avoid this in teaching that, ophthalmoscopically, *primary* atrophy is the type where there are no indications of an antecedent inflammation; *postneuritic* atrophy, where there are evidences, no matter how insignificant, of previous inflammation, including in this group neuritis, papilledema and retrobulbar neuritis; *secondary* atrophy to be an atrophy following fundus pathology—pigmentary degeneration of the retina, retinochoroiditis, toxic ambylopia, obstruction to the central artery, etc.

Doubtless this classification may appear logical to no one but myself, but I think it does clarify, in a measure, the situation for teaching purposes.

Dr. Walter R. Parker, Detroit: I agree with Dr. Jackson in that it is desirable to classify all diseases from a standpoint of etiology, whenever such a classification is possible. I am impressed, however, by the fact that the etiology of optic atrophy is definitely known in a very

small percentage of the cases, and even a presumptive diagnosis of the cause is possible in only about one-half of the cases. Until such time as we are able to classify a larger number of these cases on an etiologic basis, it seems to me advisable to agree on some classification that will include as many cases as possible, selected because of demonstrable changes. At present, there is often some obscurity in published reports due to confusion of terms. The ophthalmoscopic appearance of the optic nerve and surrounding structure suggests the classification of these cases into one of two groups, namely, simple or consecutive. Those classified as simple might be primary or secondary, that is, one might be unable to determine definitely whether the destructive process originated in the nerve or not. On the other hand, all consecutive cases must be secondary. As the etiology becomes better known, the specific type of atrophy could be included as Dr. Jackson suggests.

Dr. Edward Jackson, Denver (closing): It seems to me that Dr. Parker's division into primary and consecutive atrophies is a very practical clinical division, but our diagnosis of optic atrophy should not be based entirely on the ophthalmoscope, and probably in the near future will be based less on the ophthalmoscope than it has been in the past. Dr. Taylor, of Moorefields, pointed out years ago that the diagnosis of optic atrophy was not an observation but an inference—an inference drawn from three things: the appearance of the nervehead with the ophthalmoscope; the central acuity of vision; and the field of vision. Probably the central acuity should be placed with other disturbances of the field of vision. But the field of vision should enter largely into the diagnosis of the existence of optic atrophy, and in the future may still more enter into the discrimination of the cause of optic atrophy. I think the field of vision will show that atrophy which is due to vascular changes in the optic nerve begins at the periphery. We can hope to find by accurate observation of the field of vision, at a relatively early date, indications of the cause of some obscure atrophies now generally classed as senile changes. I want to point out the possibility of differentiation of optic atrophies, by careful testing of the fields in these cases.

AN IMPROVED TECHNIC FOR IRIDECTOMY FOR GLAUCOMA.

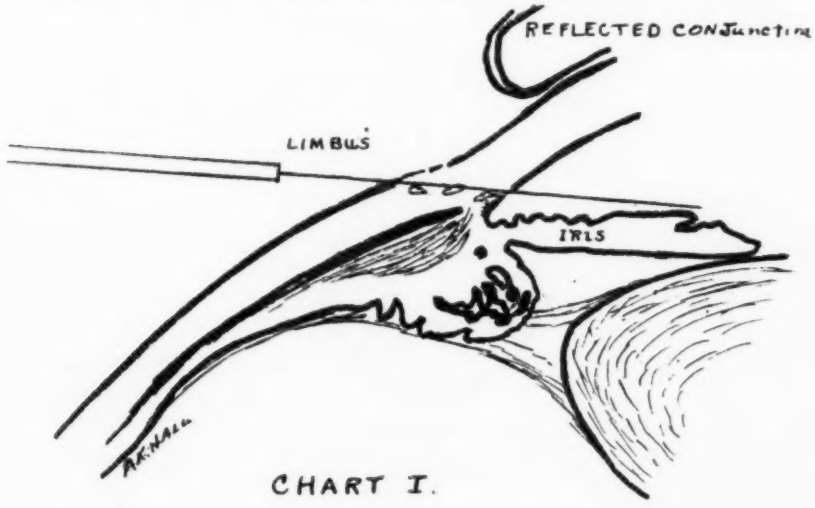
GEORGE FRANCIS SUKER, M.D., and BEULAH CUSHMAN, M.D.

CHICAGO, ILL.

It is not the purport of this paper to discuss pro or con the indications for any of the modern operations for glaucoma of any type or character, but to limit the remarks and discussion to iridectomy when this is the choice of operation for glaucoma.

From a rather large experience, we have concluded that the broad and deep iridectomy is the one operation which yields prompt and lasting reduction in tension, and maintains the visual acuity and fields as safely and definitely as any operation. By a broad iridectomy, we mean a liberal one. By a deep iridectomy, we mean one that reaches to the very root, even up to, if not attacking the peripheral ciliary mechanism.

The operation about to be described advances no strikingly new principles, but employs several principles used in other operations for glaucoma. Unless the technic is carried out in every detail as described, this operation does not offer



anything new or any better results than the "usual" iridectomy.

DESCRIPTION OF OPERATION.

1. Make a curvilinear conjunctival section about midway between the limbus and the superior rectus insertion, with the convexity toward the cornea—a section similar to that for an Elliot trephine. With such a flap, one secures a better coaptation and less retraction downward (Chart I).

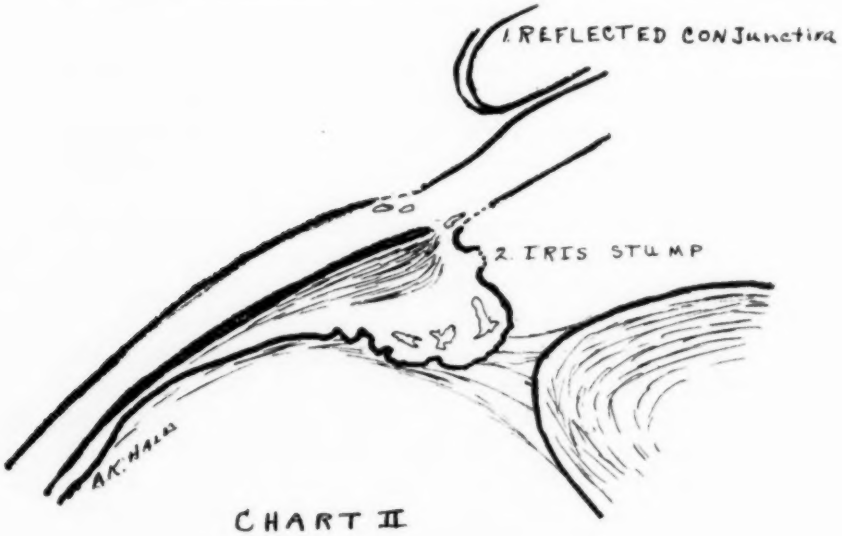


CHART II

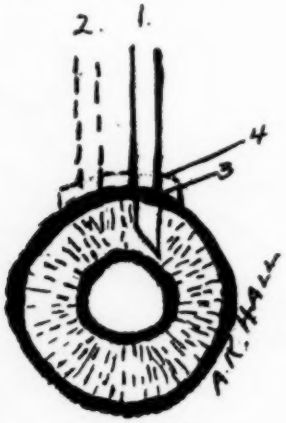


CHART III

1. Point of entry. 2. Point of exit. 3. Incision 6-8 mm. long (serrated). 4. Entry point 1-2 mm. back of limbus in sclera.

2. Catch flap firmly with broad fixation forceps.

3. Dissection of this conjunctival flap close down to sclera, preferably with a knife.

4. Extend dissection well over and beyond limbus, but not splitting cornea (Chart II).

5. The width of this dissection at limbus should be ample enough to give at least 6-8 mm. of free limbus (Chart III).

6. With cataract knife, enter anterior chamber perpendicularly at a point at least 1-2 mm. beyond the limbus, at either end of exposed area. The knife blade is anterior to the iris surface and penetrates into the anterior chamber about 1 cm. (Charts I and III).

7. Complete the section by an upward saw-draw cut along this retrolimbal line to a point opposite the entry, making a section of 6-8 mm. long. This is virtually an anterior sclerotomy section.

8. This makes a shelving serrated cut, with an inclination upward and backward, practically within the sclera and through the scleral spur, thus being very near the root of the iris (Charts I and III).

9. With forceps, grasp iris midway between the pupillary edge and periphery.

10. Draw iris downward and forward at one angle of the section, in a direction toward the cornea or over the limbus.

11. Make a small nick into iris close to upper edge of section; then for a firmer hold, if necessary, grasp the iris back of the first forceps.

12. Continue drawing the iris downward, and again nick it.

13. Continue this drawing and nicking until the opposite end of the section is reached, when the iris is gently drawn in the opposite direction and entirely severed. This latter point avoids iris incarceration at the angle of the wound.

14. In nicking the iris with a fine iris scissors, attempt to have one blade of the scissors skirt underneath the upper scleral edge, so as to pierce the iris in cutting. Repeat this in making each subsequent nick.

15. Replace conjunctival flap, and with the spatula stroke same definitely and firmly into place for good coaptation. Sutures are seldom indicated.

16. Instil one drop of 1% atropin, if you wish.

17. Bandage eye as is your custom.

18. Within 36-48 hours, if feasible, dispense with occlusive bandage.

Some advantages of the operation are:

1. Conjunctival flap.
2. Cicatrix not in corneal tissue.
3. Iridectomy involving root.
4. Broad iridectomy.
5. A serrated sclerocorneal section favoring filtration scar.
6. Prompt healing.
7. Tension reduced, and seems to remain so.
8. Miotics have not been necessary afterwards.
9. Late infection reduced to a bare possibility.

The essential steps to be closely followed are:

1. The making of the conjunctival flap.
2. Entering the anterior chamber at a point 1-2 mm. from the limbus in the sclera.
3. The upward slant of a serrated scleral section.
4. The drawing of the iris downward over the cornea, thereby allowing some iris pigment to adhere to the section, which favors the formation of a filtering cicatrix.
5. The nicking of the iris underneath the scleral edge.
6. The successive traction downward of the iris and the successive nicks, thus not tearing the iris and favoring the above deposition of pigment.
7. Some iris pigment must remain attached to the scleral wound, for reason above suggested, for favoring a filtering cicatrix.

When the operation has been performed as described, and the eye is examined within 24-48 hours, you can readily see the suspensory ligament and edge of lens in the coloboma. The pillars are absolutely free and never incarcerated, and you have a free filtering angle. In some cases, even the ciliary organ can be seen.

It is quite advantageous to precede the local anesthesia by one or two injections of scopolamin (gr. 1/150) and morphin (gr. 1/6). This anesthesia seldom if ever fails to relieve any operative pain and insures rest thereafter.

In cases with an obliterated anterior chamber, the scleral section can be made in a manner similar to the method in cyclodialysis.

From our description of the operation it is very obvious that it is the technic and not so much the principle that is different from other iridectomy operations.

The description for this technic is based upon ten recent cases, which include hemorrhagic, absolute, simple chronic, inflammatory, secondary glaucoma and buphthalmos. We did operate in this manner, but never reported it, at the time Elliot's trephining became popular, which we then adopted, and did not again revert to the operation described until about a year ago.

We wish herewith to thank Dr. Alice K. Hall, Chicago, for the several very illuminating illustrations.

DISCUSSION.

Dr. Lee M. Francis, Buffalo: This discussion must of necessity be based entirely upon theoretic grounds, because I have not had the opportunity of doing the operation as described by Dr. Suker.

The technic provides for an ample conjunctival flap, which, because of its design, should promptly go into place.

To those who feel that some of the good results of iridectomy may be due to a filtering scar, the substitution of the cataract knife for the keratome will appeal. Certainly, a keratome wound heals cleaner and more promptly than one made with any other cutting instrument. The sawing motion necessary to complete the incision makes for a more irregular wound, and one more apt to become filtering. It would appear from the description, that should the aqueous be lost upon first entering the anterior chamber, the completion of the incision would be somewhat difficult because of the bellying forward of the iris, and also there might be some danger of wounding the lens. The latter danger might be readily overcome by making the counterpuncture, as advocated by Colonel Smith.

Dr. Suker's technic provides for a shelving lip of scleral tissue. I see no reason why this technic might not readily be turned into a filtering scar operation similar to the Lagrange, by the removal of a part of this wedge.

The most important part of the procedure appears to be the method of stretching the iris in front of the cornea, and trimming the iris to the root by successive snips of the scissors.

We believe that the successful iridectomy for glaucoma is the one which is deep and broad, especially the former. Dr. Suker's technic appears to insure this most important feature.

The single case which I have seen was one most unfavorable for operative attack—that of a hemorrhagic glaucoma with a tension over 70 Schiötz. Ten days after the operation, the tension had been reduced to 50, the patient was free from his excruciating pain, the wound had healed nicely, and there was relatively little pericorneal congestion. A deep and broad iridectomy was very evident.

Dr. H. W. Woodruff, Joliet, Illinois: I have had some personal experience in the last two years with the operation of deep iridectomy for

glaucoma, the method followed by Török of New York and described by him something over two years ago. The making of the scleral incision is absolutely necessary in order to be able to cut the iris off at its root. Dr. Suker's chart, which is being passed around, does not indicate that his incision is as deep or as far back in the sclera as it really should be. I have had some experience in experimental work on the eyes of pigs and kittens, and it is an exceedingly difficult matter to place the incision just where you desire, that is, to get it back far enough. The measurements are so extremely small in the angle of the anterior chamber, that it is not always easy to place the incision where you wish it without injuring the iris.

I am particularly pleased with this operation in the type of glaucoma known as the chronic simple variety, the type of glaucoma in which iridectomy has not often given satisfaction. This fact has given rise to the operations of external drainage, such as the Elliot and the Lagrange operations.

Last Monday I showed a case to some of the visiting doctors, a case of chronic simple glaucoma in a young woman upon whom I had operated two years ago. It was the first patient upon whom I had performed this operation. She had had double iridectomies, which reduced the tension well within normal limits—15, 16 or 17—and it has remained constantly within these limits for two years. Also, in this case there was no sign of external drainage. The wound seemed to be absolutely closed, with no sign of filtering cicatrix. I have operated on 20 of these cases of chronic, simple glaucoma by this method, with 100 per cent results, if I exclude two cases lost from choroidal hemorrhage. These cases were of varying degrees of loss of vision and of tension. Some of them were blind; some had very little vision; some had normal vision; but all were of that type—no inflammatory symptoms whatever. Following the operation, some did show distinct signs of external drainage; some of them showed no signs at all. So I am quite convinced that you secure good results in one of the two ways, or both.

I do not exactly understand Dr. Suker's nicking method in incising the iris, but the essential features of this method of Török are the deep scleral incision with a cataract knife, going as far back with the puncture and counterpuncture as possible, so that the point of the cataract knife is placed in the extreme angle of the iris, then the incision continued through sclera and conjunctiva, so that a very broad flap is made. The iris and ciliary body are separated from the sclera with the spatula, then a drawing of the iris away from its root, but with the iris forceps placed horizontally instead of vertically, as we ordinarily do.

One additional point about the making of the incision. To make a peripheral incision with a cataract knife is not so easy as making the cataract incision, because of the tendency of the globe to rotate. It is recommended by Török that double fixation forceps be used. I find Dr. Ewing of St. Louis has a forcep which answers the purpose very well and prevents rotation of the globe. I have performed this operation in many varieties of glaucoma with varying results. The only instance in which I would be inclined to say positively that the operation should not be used would be in buphthalmos cases.

Dr. Charles E. Walker, Denver: The operation that Dr. Suker has

described is very similar to an operation which I have done a great many times in the last 25 years, and I want to endorse his statements.

There are two points I would like to bring out. In the case of glaucoma, as a general rule, we have the iris forward. It is very awkward to make an incision without engaging the knife in the iris. That can be obviated at the time of operation by making a posterior scleral puncture. I follow the method of Priestly Smith of Birmingham. That is:

Have the patient look down and in, and as the patient as a general rule is under a local anesthetic, you can slide the flap on the conjunctiva, placing the Graefe knife posterior to the ciliary body, the point towards the center of the eye, piercing the sclera posterior to the ciliary body, passing it to the center of the eyeball. Now turn it slightly and withdraw. I generally ask the patient if he suffers any pain. In glaucoma it is very difficult to get the patient under an anesthetic, and sometimes it is necessary to use a general anesthetic on that account. Take a small Graefe knife called a Lang knife, pass it near as possible towards the center of the anterior chamber, clearing the iris and making an incision in the cornea about one-fifth. You cannot in a glaucoma operation, even by making a scleral puncture, get back into the scleral tissue to make the first puncture. You must clear the iris. Then, as you pass across the anterior chamber, clear the iris, turn it and come out, keeping the blade clear of the iris, make your puncture, cut it upwards, turning the blade of the knife back and coming out in the sclera, and making a large conjunctival flap. With the iris forceps, pick up the iris at either angle, make a snip; pull the iris to the opposite side, tearing it well at the root, and make your second cut, making a large iridectomy. This, of course, will form a cystoid scar in the healing. By turning the knife backward, you separate the angles of the eye so there is filtration through these angles.

Dr. George F. Suker, Chicago (closing): The charts shown are simply diagrammatic, and it is hard to get them accurate.

I started to do this iridectomy some fifteen years ago, but gave it up in a way because the trephine appeared and I was enthusiastic about it, and I still am in certain cases.

I do not believe that the incarceration of the iris is a very scientific mechanical operation. Iris pigment adherent to the scleral wound edges favors and produces a filtering cicatrix as efficiently as incarcerated iris—and that without any untoward after effects.

The difference between this operation and Török's, which I know quite well, is that I do not use a spatula to loosen the ciliary body. I do not think that is a very safe procedure. The coloboma that is left is very large in many instances, and you can readily see the ciliary body, the suspensory ligament and the lens periphery through it. If the anterior chamber is so shallow that you are afraid to enter with a cataract knife, bear in mind that the posterior chamber is relatively deep, and you can still cut across the sclera as you do in cyclodialysis. The loss of aqueous is relatively slow.

I am glad that Dr. Walker has had a similar experience and has been doing a like operation for some time. It appeals to me for several reasons—first, because of its simplicity; second, because of the accuracy with which you can make the scleral section; and third, because a serrated wound favors filtration. It is seemingly a combination of the best steps of various iridectomy operations for glaucoma.

OSTEOMYELITIS OF THE FRONTAL BONE AS A COMPLICATION OF FRONTAL SINUSITIS

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Osteomyelitis of the frontal bone as a complication of frontal sinus suppuration is a relatively rare condition. A search through much of the American and European literature for thirty years back discloses reports of a total of fifty-five cases, thirty-seven of which ended in death. The nature and etiology of some of those are questionable owing to incompleteness of the reports. Of this total number, twenty-eight may be classed as occurring as a complication of chronic suppuration of the frontal sinus, twenty-one of which resulted in death and seven in recovery. Out of these twenty-eight chronic cases, all of which were operated, in but eight of the number was osteomyelitis discovered at the time of the first operation, resulting in four deaths and four recoveries; and in twenty the osteomyelitis developed subsequent to operative attention for the sinusitis, and subsequently required operation for the osteomyelitis, resulting in seventeen deaths and three recoveries. Gerber[1], in his analysis of cases in 1909, found a similar proportion of cases of osteomyelitis developing subsequent to operative interference, and was led to make the remark, "What operative error is responsible for this grave sequel we do not know, for it befalls all, old experienced operators as well as tyros."

Of this total of fifty-five cases, twenty-seven occurred as a complication of acute suppuration of the frontal sinus (all of which were operated), resulting in sixteen deaths and eleven recoveries. In eighteen of the acute cases the osteomyelitis was discovered at operation, resulting in ten deaths and eight recoveries. Nine of the twenty-seven acute cases developed osteomyelitis after the first operation, and of these six died and three recovered. Three of the acute cases were what may be called spontaneous or fulminating osteomyelitis, occurring coincidentally with the development of the acute

frontal sinusitis, all of which resulted in death. These spontaneous cases occurred without evidence or history of previous nasal trouble, though it is quite possible and even probable that most of them may have occurred as a direct result of lighting up a latent infection, of which previously there had been no discoverable evidence. At all events, while osteomyelitis of the frontal bone under any circumstances is a comparatively rare and serious affection, as evidenced by the limited number of cases reported in American and European literature, those that develop spontaneously or fulminantly are still rarer, with death resulting in all cases reported, and it is one of the latter, with recovery of the patient, that I desire to report:

CASE REPORT.

Patient, D. I.; age, twenty; motor inspector; referred to me by the General Electric Company on August 16, 1924. Previous history negative. Has had neither serious illness nor nasal trouble. History: Late in the afternoon of August fourteenth, received a comparatively mild blow from a screw which slipped and flew from a screw driver, striking patient at the root of the nose just beneath the left eyebrow, but without producing a visible wound of the skin surface or even evidence of a bruise. Four or five hours after the accident, there was some localized headache in the left frontal region, and the following morning, about fourteen hours after the accident, there was considerable swelling at the root of the nose between the eyes, and an increase in the general left sided headache, for which latter the family physician prescribed sedatives. On the following day, both the pain and the swelling had increased and the patient had two degrees of fever.

On the third day, when I first saw the patient, the symptoms were more marked, and the edema involved the left upper eyelid and extended to a point on the forehead about one inch above the root of the nose. There also was considerable congestion and chemosis of both the ocular and palpebral conjunctiva. Suspecting a localized infection of the orbit, a deep incision was carried into the orbit just above the inner canthus, but not penetrating the bone, with the evacuation of a small quantity of serosanguinous fluid. The wound was injected with five percent alcoholic mercurochrome solution and hot moist compresses ordered.

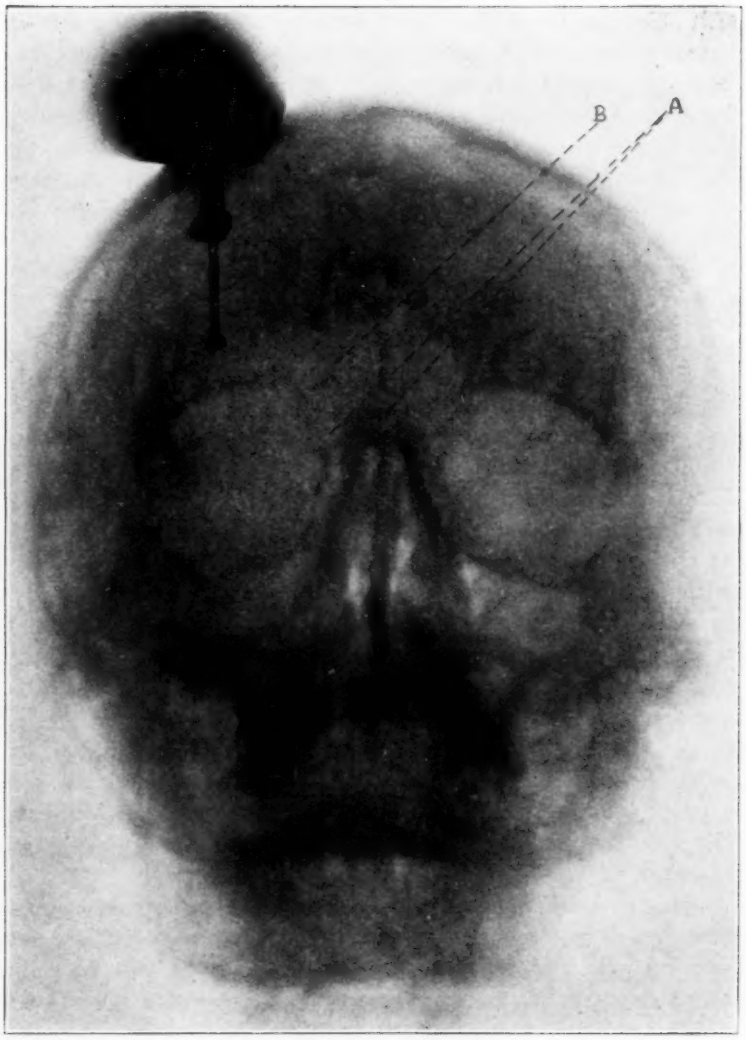

Figure 1. Condition, August 18, 1924: *A*, broken down bone with fistula between; *B*, cloudiness of the frontal sinus.

On the following day, less than four days after the injury, all the symptoms and manifestations were more marked, the edema extended upward to nearly the hairline and over the left temple, and the eye was closed from edema of the surrounding tissues. The patient was very nervous, temperature 100.2°, pulse 120. Thirty grains of sodium salicylat was administered intravenously, and the patient sent to the Fort Wayne Medical Laboratory, where Dr. E. M. VanBuskirk made a roentgenographic examination.

On the following morning another deep incision at the root of the nose, just beneath the eyebrow, encountered softened bone and an abundance of pus. At this time the nose was free from any evidence of sinus involvement, but later in the day the roentgenologist's report was received and indicated that the left frontal sinus contained pus, and the anterior bony wall was involved. The sinus was then opened intranasally, a good sized opening being established, and a considerable quantity of pus evacuated through the nose. At that time there was a large boggy swelling involving all of the tissues from the inferior bony orbit upward to the hairline, and from the median line of the forehead nearly over to the left ear. Dr. B. W. Rhamy, of the Fort Wayne Medical Laboratory, reported that the pus in the frontal sinus showed a pure pneumococcus infection. The patient was sent to the Methodist Hospital (previously he had refused to go to the hospital), the wound was irrigated with five percent alcoholic mercurochrome solution, and steaming hot compresses ordered. Dr. Rhamy reported negative Wassermann, and white cell count of 12,400.

On August 24, after a bad night, with temperature 104°, pulse 130, the incision at the root of the nose was enlarged, with the removal of several pieces of softened bone and a considerable quantity of pus. Incisions also were made deep into the orbit above, and to the outer side near the external canthus, pus being evacuated from both incisions. The patient and relatives refused a radical operation.

From August 22 to August 30, there was a free discharge of pus from the three wounds that were kept open with drainage, and there was a gradual decrease in the edema and a drop in the temperature to nearly normal. Except during the first few days, the patient had experienced little or no pain. The patient insisted upon leaving the hospital on September

1, at which time there was a free opening into the left frontal sinus at the root of the nose, and another small fistulous opening into the sinus through the orbital plate, immediately above the eyeball; and with a probe considerable softened bone could be felt in the surrounding area. During the next six weeks, or up until October 27, pus exuded from the three fistulous openings almost constantly, and occasionally a sequestrum of bone would present itself where it could be removed with forceps. The later roentgenograms showed extensive involvement of the frontal bone, including the bony covering of the dura. The pus still showed almost a pure pneumococcus infection.

Finally, the patient was induced to reenter the Methodist Hospital for a radical operation, which was performed on

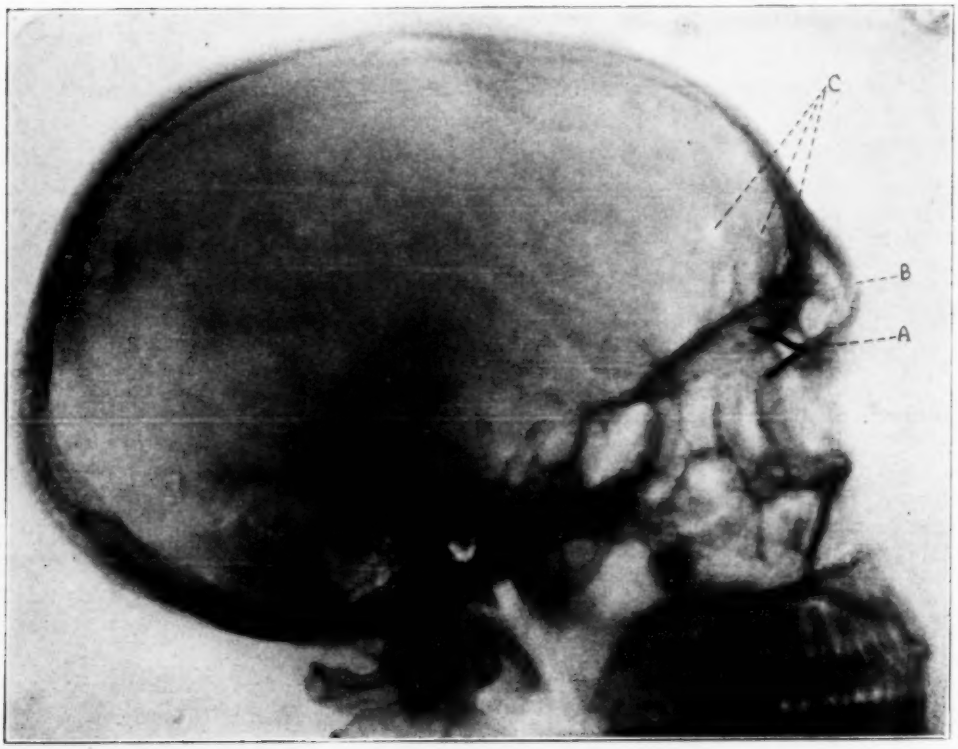

Figure 2. Condition, September 29, showing bone tissue breaking down: *A*, fistulous opening through the bone through which has been passed a lead style; *B*, fistulous opening through the bone; *C*, areas of bone destruction.

October 27, nearly two and one-half months after the onset of his trouble. A wide incision was made through the eyebrow from the median line well over on the temple, through which the osteomyelitic bone was removed by curetting, necessitating considerable exposure of the dura. Fortunately, a good portion of the orbital rim seemed to be unaffected, and, in consequence, the resulting deformity was not great. Much of the bony wall had a worm eaten or honeycombed appearance, while some of it was merely softened to the extent that it could be wiped out with a gauze sponge. Wherever the osteomyelitic process was found, the curettement was carried down to healthy tissue. The wound was swabbed with five percent alcoholic mercurochrome solution, left open, and rubber tube drainage provided at the root of the nose. The right frontal sinus was not affected. With the exception of three or four minor curettements and the discharge of several small sequestra of bone during the succeeding few weeks, the patient went on to a satisfactory recovery. Subsequent X-ray plates disclosed a considerable amount of regeneration, and up to the present date he has remained well.

The interesting feature in connection with this case is that the osteomyelitis was of such sudden development, and that apparently the inciting cause was trauma, with perhaps injury of the outer bony wall of the frontal sinus at the root of the nose, even though there was no discoverable break in the skin surface. The bone involvement may have come from a latent frontal sinus infection, even though we have no means of definitely proving the route traveled by the infection. However, we know that the infection may travel by two routes, first, by direct contiguity of structure, and, second, by the vascular route, in both of which instances there is hyperemia of the periosteum, going on to the formation of hemorrhagic foci and the production of subperiosteal abscesses. It is entirely probable, therefore, that there was a latent infection in the frontal sinus which required only a lowered resisting power and the inciting trauma to start a pyogenic process in the bone structures.

That osteomyelitis of the frontal bone is serious is evidenced by the death rate already quoted. The disease, as pointed out by Luc[2], Knapp[3], Schilling[4], Tilley[7], Gerber[1], McKenzie[5] and others, is an inflammation invading the frontal bone over a wide area, very difficult to control, with the de-

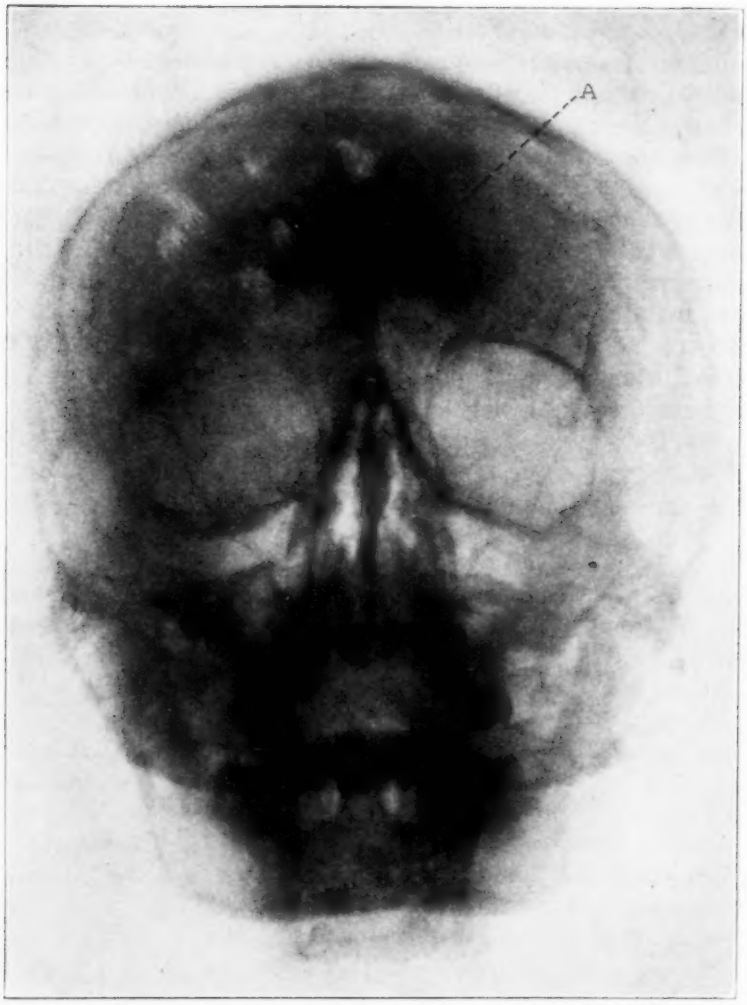

Figure 3. Condition, October 10, just before operation; *A*, extensive breaking down of the walls of the frontal sinus and patches of osteomyelitis of frontal bone.

velopment of osseous abscesses and destruction of the bony tissues. As McKenzie[5] well says, once osteomyelitis has set in, the only chance of saving the patient's life lies in the prompt and entire removal of the diseased bone. The sacrifice of large sections of the bony cranium need arouse no apprehension, for if the case recovers the defect will be made good very largely by osseous regeneration. As the history of numerous cases reported will show, marked remissions may occur which lead to a false sense of security. Six to twelve months should elapse before the patient is released from control. We seldom deal with an unmixed infection, though an analysis of numerous cases presented indicate that pneumococcic and streptococcic infections give the most violent course, are hardest to control, and most likely to end in a fatality.

Concerning treatment, I believe that most will be accomplished by the open treatment of the wound after thorough removal of all of the diseased bone. Ventilation as well as drainage aids the recovery of these pyogenic processes, and I believe that the penetrating and antiseptic effect of some of the dyes, particularly mercurochrome soluble 220, used locally, aids in limiting the progress as well as destroying the infection. I have little faith in vaccines, ever though they are supposed to raise the resisting power by building up the tissue defences against the inroads of the infective process, though I can see no harm in their judicious use. If of value, they would exert their beneficial effect most *after* the complete removal of the diseased bone.

To quote Dan McKenzie[5], who in 1913 published the most extended article on the subject in the English language, "Osteomyelitis of the skull prior to the antiseptic era was a common consequence of open injuries to the cranial bone, and while the case histories and the early books on surgery indicate that the disease must have been a familiar experience with the surgeons of the seventeenth century, its true nature and even its most striking feature, the association of the paracranial abscesses with extradural abscess, remained unknown until Percival Pott, in 1760, first drew attention to what came to be known afterward as Pott's Puffy Tumor." That these paraosseous abscesses depend upon a disease of the bone was discovered first by von Bergmann and Lannelongue[6] in 1879, but it was not until nearly twenty years later, or until

Figure 4. Condition, December 12, after operation: *A*, obliteration of part of walls of sinus; areas of extensive curettage and exposure of dura. Considerable bone regeneration has occurred since operation.

1897 and 1898, that the occurrence of osteomyelitis as a complication of nasal sinus suppuration was detected first by Tilley[7]. Before that event, osteomyelitis of the cranial bones generally had been ascribed to some systemic malady of the septicemic order, such as measles, typhoid fever, erysipelas, carbuncle, etc. Since then, the cases reported in the literature have been for the most part referred to nasal sinus or aural suppuration.

McKenzie[5] very frankly admits drawing freely from the writings of Schilling[4], Gerber[1], Luc[2] and Knapp[3], whose articles are especially comprehensive and instructive and whose observations and opinions are repeated practically verbatim in nearly all of the articles that have appeared since.

Schilling[4] reports four cases where osteomyelitis of the frontal bone followed frontal sinusitis, two being his own cases, another observed by Körner, and a fourth observed by Laurens. The history of the author's own two cases is given in great detail, with all the variations in his technic of treatment. He considers the affection as rare, and comments on the preponderance of occurrence in the female sex and the fact that the disease is not only more frequent but more rapidly progressing between the ages of fifteen and thirty years, a fact which the author attributes to the great activity of development of the skull bones during adolescence and early adult life, and he quotes Graf von Spee to the effect that "in general, a rapid increase in the thickness of the skull is observable toward the end of childhood, at the onset of puberty, and indeed it is chiefly under the influences operating at this period that the formation of spongy tissue in the bone, the socalled diploë, largely takes place." Schilling noted that the infection seems to follow both acute and chronic frontal sinusitis, and mentions three cases in which the retention of pus preceded the onset of the osteomyelitis. In another case the osteomyelitis appeared eight days after the suppuration had suddenly ceased, and he noted that retention of pus was very generally shown clinically; and at operation he was unable to decide whether or not any definite bacterium played any part in the extension of the suppurative process to the bone marrow by way of the mucous membrane, and concluded that no particular form of infective organism could be considered as the causal factor. A fairly comprehensive abstract of the main discussion in his paper is as follows:

Theories Concerning the Manner by Which the Infection Extends are: (a) By continuity after the destruction of the mucous membrane covering the walls of the sinus, first the wall itself and then the diploë become infected; (b) indirect infection through the capillaries or veins.

(a) Relation of the position occupied by the diploë to the neighboring accessory sinuses. In sagittal sections of cranial bone, no diploë usually is seen in the anterior cerebral and inferior wall of the frontal sinus. Only where cerebral and anterior sinus walls meet does the sinus border directly on the diploë of the squamous portion of the frontal bone, at which point it is separated only by a thin ridge of compact bony substance. In a few cases, the anterior sinus wall, and still more rarely the cerebral wall, contained diploëtic substance in direct relation with that of the frontal bone. The condition was noted to vary even in different portions of the same sinus. Therefore, the chance of the extension of a suppurative process varies in different individuals; and for the same reason the danger of exposure of the diploë to infection by operation is much greater in one patient than in another.

(b) Infection may take place through the small vessels of the mucous membrane and periosteal covering of the accessory sinuses, as those of the mucous membrane and bony walls of the sinus are in communication with those of the cranial bones. The infection may be borne by the blood stream or travel along the outside of the vessels as they pass through the bone.

When the infection has reached the diploë, conditions favoring extension are (a) the loose structure of the marrow and spongiosa, and (b) their rich venous supply. The first depends upon age and individual condition of the patient—the modification of the diploë at the onset of puberty, or as frequently happens as age advances, thickening and increase of bony solidity, or atrophy and shrinkage of the diploë. The first condition would favor rapid increase, the second would have a retarding effect. As the blood vessels of the different bones are in close relation through an extensive network of canals—more especially those of the diploë—, in certain cases it was plain that thrombosis of veins took place, thus favoring extension of the suppurative process. It is, however, easy to confuse an extension of infective foci from an original thrombophlebitis with a direct extension through the diploë.

Periosteum and Dura: Generally the disease process showed extension in a collection of pus on both sides of the affected bone; one side being subperiosteal and the other an extradural abscess. These seem always to develop coincidentally, never one appearing alone. After destruction of the periosteum occurs, there will be abscess formation of the soft parts, with swelling and edema. Operation shows denudation of the bone with more or less alteration in its structure. On the dura, a granular cushion of varying thickness quickly forms, which replaces the pus and erodes the bone superficially. No fistula will be found which could have acted as a medium for the conveyance of infection. Microscopically, in the case of a child of nine months, abundant vascular conduction was noted, which the author explains by the action of the suppurative process in dissolving the sutures between the bones, thus rendering communication easier.

Macroscopic Changes in Diseased Bone: Discoloration: In the early stages, hyperemia of the diploë with foci of pus, the consistency of the bone being softened. After ten days' swelling, the diploëtic spaces were filled with pus and granulations; after four weeks, the bone was discolored all through. Later changes consisted in the filling of the diploëtic spaces with granulations, with partial breaking down of the first fortifications of resistance of the tabula externa and interna, the granulations being separated into unrelated centers. Sequestra had been cast off from the frontal, parietal, temporal and occipital bones.

Histology: In the earliest stage, one sees fairly well marked medullary spaces filled with small celled infiltration. The bony walls are smooth and unchanged, though here and there are signs of lacunar resorption. The surface of the dura is rough and detached, showing lacunar formation. In the vessels, infiltration of pus is visible, noticeably in those running into the dura and periosteum. In the later stages, also, the medullary spaces are full of small celled infiltration, containing a network of connective tissue fibers and vessels. The walls of these spaces show still more lacunar bone resorption. Isolated bits of bone, small sequestra, are imbedded in the granulation tissue. Regenerative processes are in evidence also, as one can detect the beginning of new formation of bone. Where the regressive processes predominate, there are

great numbers of lacunae and osteoblasts, with only small isolated bits of neoplastic tissue. Once infection is established in the cranial bone marrow, the process can be checked by nothing except operation.

In the fourth and fifth cases (author and Knapp) the osteomyelitis extended from the frontal sinus to the occipital bone; in the seventh and eighth (otitic cases), from the temporal bone to the parietal and frontal bones. The time occupied varied from a few days to months or even years. In the protracted cases operation gave a temporary respite.

The dura is always more seriously affected by the suppuration. Though at first highly resistant, if this resistance be overcome, the suppuration may extend to the pia and arachnoid brain substance, and the large cerebral sinuses. In case five, the thrombosis of the sinus transversus probably was due to extension of extradural or parasinus abscess directly upon the sinus wall, followed by phlebitis of the cerebral sinuses, the sinus cavernosus longitudinalis superior, and involvement of the other side and the entire jugular vein. Killian and others report corresponding abscesses between the periosteum or dura and the bone, suggesting the possibility of foci of osteomyelitis in the bones. In Schilling's second case, the osteomyelitis started from the frontal sinus, wandering through the entire side of the head, attacking the middle ear, the mastoid process, labyrinth and finally, the meninges.

The initial symptoms are local pain, usually due to the suppuration, edematous and painful swelling, associated with extension of the inflammation, rise of temperature, general malaise, restlessness, chills, and sometimes nausea and vomiting. In one case there was a partial one-sided facial paralysis, possibly due to the extradural abscess.

Operation for the removal of the diseased focus sometimes leads to complete recovery. More often, however, there will be only temporary relief, with the appearance of a new focus, either adjacent to the previous one, or in what was apparently previously sound tissue. A second operation may bring cure, but more commonly new foci continually arise, with progressive shortening of the pain free intervals. Changes in the fundus oculi were noted in some cases. Death usually closes the scene by disease of the brain and meninges, or by pyemia resulting from infection of the blood stream through the sinuses.

Guisez[8] confirms the observations of Schilling, Luc and others concerning age and sex of those subject to osteomylitis of the frontal bones, and regards previous infectious diseases as a uniform contributive factor. Epidemic la grippe and scarlet fever are contributive factors. Other predisposing causes cited are a generally enfeebled condition, albuminuria and diabetes, overwork, certain dyscrasias which render it difficult for some individuals to resist infection, malnutrition and the debilitating effects of severe fevers which render the subjects peculiarly liable to infection during a protracted convalescence. It is in chronic rather than acute sinusitis that the diffusion of the infection to the adjacent osseous tissue takes place, and Guisez says that the process is favored by retention of secretion in the sinus; though he admits that in practice it is hard to tell whether one has to deal with an acute attack or the relighting of an old chronic process, as it is well known that the sinusitis may be latent for long periods and even go unrecognized for years.

A condensed abstract of Guisez's discussion is as follows: Osteomyelitis frequently follows operative interference, and even in those cases where apparently every diseased bone cell has been removed, it will be found that the diploë already was compromised so much as to make it impossible to abort the infection. In few if any cases do we deal with an unmixed infection, though the staphylococcus has been cited as the chief offender. Even granting the specificity of any one microbe, we still must admit that the epidemic character of the infection, the soil upon which it is implanted, the powers of resistance retained by the patient following his previous infection, must be rated as of greater importance than the development and diffusion of the process. The disease is so rarely reported, that only a few cases are on record. It is an inflammation invading the frontal bone over a wide area, very difficult to control, rapidly infecting all of one side of the cranium, and even passing to the other side. The diploë and the external and internal periosteum all participate, but it is in the region of the diploë that the phenomena of suppuration are manifested and developed. The process confines itself to the areas of spongy tissue confined in the invaded osseous structures.

Reviewing the work of Breschet and of Schilling, Guisez reaches the conclusion, that (1) In all young subjects up to the age of eight or ten years, the infection appears to progress from one focus to another (de proche en proche) by virtue of the medullary richness of the diploetic element of the bone: (2) In adults, and especially in the aged, the vascular route is that most often traversed by the infection. (3) The development of isolated foci in the bone is a process similar to that of phlebitis by metastasis, the pathogenesis being analogous to that of isolated plaques of meningitis.

At the onset of the infection, an intense congestion is observable, resulting in dilatation of the blood vessels and augmentation of the soft osseous elements. As in all microbic inflammations, considerable leucocytosis develops. This brings about a hyperemia of the periosteum, going on to the formation of hemorrhagic foci, and at the same time an inflammatory subperiosteal edema becomes evident, which rapidly denudes the bone by producing subperiosteal abscesses on both its surfaces. The internal abscess is extradural and lies between the internal periosteum and the bone itself. This process is characteristic of osteomyelitis of the cranial bones. The pus content of the abscess is thick, greenish and sticky, and loaded with pyogenic organisms. Within the substance of the bone proper it produces little osseous abscesses, distending the Haversian canals, and destroying the bony lamellae in the same manner as is accomplished by rarefying osteitis.

The point of departure in the infection of the frontal bones in frontal sinusitis is usually in the anterior wall of that sinus. Though ordinarily compact in the region of the eyebrow, under these conditions there will be a thick layer of spongy tissue over its whole extent, especially at the lower frontal protuberance. This bleeds profusely when the anterior wall of the sinus is surgically invaded, and the whole area is peculiarly liable to sepsis, offering little resistance to the extension of an infectious process.

Infection also undoubtedly travels along the veins, and though no study has yet been made of the lymphatics of this region, it is likely that they too play no small role in extending the lesions. Once established, the foci of infection rapidly spread, invasion of the sphenoid being especially to be dreaded, as it is extremely difficult to limit the process upon that

side, and the large vessels of that region soon may be gravely menaced. The remainder of Guisez' article is based almost wholly on Schilling's article and no new case reports are added.

Rõpke[8] reviews the literature and says that the discussion of the disease previously given by Schilling and Guisez are so masterly that he can do no better than quote them in extenso, which he does. Up to the period of his writing, he credits but thirteen true cases of osteomyelitis, to which he adds three of his own.

Luc[9] makes a second contribution to the subject, and includes brief abstracts of all the cases previously reported, with a second one of his own. Like the other authors, he says that cranial osteomyelitis invading the interior of the skull, originating in the frontal or ethmoidal sinus, is rare. It is his opinion that cranial osteomylitis takes place only in those subjects presenting developmental anomaly. The disease appears usually between the tenth and the thirtieth year, perhaps on account of the fact that at this period of life development and work of the brain and its envelopes is being most actively carried on. Like Schilling, this author notes that many more patients are women than men, and he quotes Breschet, who observed that in general there is greater development of the cranial diploë in females than in males, a circumstance which may account for a greater liability to the manifestation of cranial infection in women. Breschet's work on the cranial circulation as presented by Schilling is quoted at length, and the conclusion set forth that the topography of Breschet's intracranial canals governs the progression of the special form of osteomyelitis which he terms the "leaping" form. It is all along the line of these canals that those subperiosteal or extradural pus collections always are observed, which marks the serpentine progress of the suppurative process in the bone; and the author believes that in his first case, reported earlier, it was by traveling along the anterior temporal canal that the suppurative process came to form an extradural focus in the region of the paracentral lobe, evidenced at first by monoplegia, then by a hemiplegia of the opposite side, and finally soon followed by the arachnoid infection and death.

Observations made at operation or autopsy show the cranium affected by that form of osteitis which bathes the

external surface with pus, which also is found beneath this surface and even in the dura mater. The tissues will be seen to have lost their normal consistency, having become "spongy," pus exuding from a cut surface, oozing from tunnels in the bone which correspond to Breschet's intraosseous canals, as in Durand's case. The diploë may be completely destroyed and its place taken by a pus collection lying between two lamellae of compact tissue. The pathology of the cases presented by Tilley and the author before the British Medical Association is discussed at length.

The only treatment recommended by Luc is wide resection of the infected bone; he is opposed to Schilling's plan of making incisions around the outside of the suppurating area. He is inclined to favor medicinal treatment directed toward clearing the blood of infective microorganisms, which can be carried out in conjunction with surgery, if this appears wise.

P. H. Gerber's[1] contribution concerning frontal bone osteomyelitis forms Section F of a monograph which constitutes a book of 458 pages. The article begins by mentioning the names of authors who previously have written on the subject, and credit is given to the work of Schilling, Guisez, Luc and Röpke, who have directed attention to the comparative facility with which infection can travel from a diseased sinus to the bones of the skull or even to the brain itself. The author, however, questions the authenticity of some of the cases collected by these four authors, rejecting that of Thompson, and adding eleven from the literature which, with one of his own, he claims to bring the total to 29. He devotes several pages to a discussion of etiology, quoting freely from other authors, and calls attention to several cases which show incompleteness of the operation, or the fact that it was undertaken too long after infection had become established in the frontal sinus, and the medullary spaces already were involved. His own case is then described, and recovery is attributed to the radical operation with subsequent regeneration of the bone. In his own analysis of collected case reports, the author considers termination, which was in twenty-nine cases as follows: Osteitis, necrosis and perforation of the anterior wall of the sinus, thirteen cases; of the posterior wall, seven cases; of the floor, two cases; of all three walls, one case; pachymeningitis, four cases; extradural abscess, eleven

cases; thrombophlebitis, five cases, brain abscess, four cases; intradural abscess, one case; meningitis, nine cases; meningoencephalitis (one of them septic) three cases. Nine patients recovered, twenty died.

Gerber also gives an age table, showing the frequency with which the disease appears between fifteen and thirty years of age. He also refers to the involvement of both sinuses practically twice as often as in the sinuses singly.

Dan McKenzie[5] is the author of the most extended article on the subject in English, running through three numbers of the journal in which it appears. Much of it is, the author explains, drawn from the work of Schilling, Röpke, Gerber and Luc, "upon whose papers our present knowledge of nasal sinus osteomyelitis so largely depends." He questions some of the cases reported, and deducts one, that of Spicer, which proved to be syphilitic and not septic in nature, and he adds seven others from the literature, and bases his remarks upon the records of forty-eight cases in all.

A brief abstract of his paper is as follows: Osteomyelitis of the frontal bones does not differ from that in other parts of the skeleton, except that diffuse osteomyelitis of the cranium is referable in the vast majority of cases to some contiguous infective focus in the ear or nasal accessory sinuses. In none of the cases hitherto recorded, was there any information regarding the route by which the bone received its infection from the suppurating sinus. Two methods of infection are presumed; direct extension by the organisms to the osseous spaces, canalicular and medullary, of the bony wall of the sinus; and primary infection of the small efferent veins of the lining membrane of the sinus, with subsequent infection of the bone from the veins. In McKenzie's opinion, the former is the more important, but it is obvious that both methods may act in combination. The author is inclined to minimize the part played by the disposition of the diploë of the frontal bone in relation to the sinus, so emphasized by the Continental authors, who contend that the proximity of the diploë to the diseased cavity or its surgical exposure, lays it open to infection. While he believes that the infection may be able to spread more rapidly once the diploë has been reached, he holds that diffuse osteomyelitis is not limited in its spread to diploic bone only. As the disease spreads, it

involves the thin hard bones of the ethmoid, lacrimal bone, nose and orbit, and does not spare the frontal eminences, where the diploic tissue is attenuated or altogether deficient.

If the infection reaches the interior of the veins, its spread undoubtedly will be faciliated greatly. But in the adult, the skull is, for all practical purposes, a continuous bone, and the spread of infection is not limited by an impassable barrier, whether it is by dense bone or by the veins. The disease usually tends to spread upward, but this is not invariable, and a number of the cases show invasion of the nasal and orbital bones from above, while extension from the squamous to the petrous portion of the temporal bone is by no means uncommon. Gerber's table is cited in regard to local extensions and complications, and inference drawn that the danger zone lies toward the base of the cranium, where so many natural portals pierce the cranial fortifications. Septic thrombophlebitis has been found to affect the superior longitudinal, the cavernous, the circular, the petrosal and even the lateral sinuses, due to extension of the thrombosis from the diploic veins. The venous sinuses at the base of the cranium are usually infected by way of the ethmoidal and orbital veins.

Age table from Gerber's, Luc's and McKenzie's collections:

1-10 years of age	2 cases
11-20 years of age	8 cases
21-30 years of age	18 cases
31-40 years of age	4 cases
41-50 years of age	3 cases
51-60 years of age	0 cases
Over 60 years of age	1 case

Schilling's theories in regard to age are discussed by McKenzie; also Gerber's list of possible predisposing conditions. Of the last, "operation on the frontal or maxillary sinus," the author has several observations to make. Of forty-one cases "deemed valid for the purpose," twenty developed osteomyelitis after operation, while in twenty-one it appeared spontaneously before any operative intervention. All the postoperative cases died, while seven of those which were of spontaneous origin recovered after appropriate surgical treatment. As osteomyelitis is a septic disease, we may suppose that injudicious bruising of the bone edges by the forceps, prolonged

exposure at the operation, careless aseptic technic, and above all perhaps, inadequate drainage of the wound area, will predispose to its occurrence.

McKenzie says that we have little etiologic knowledge of spontaneous osteomyelitis, except that it is more apt to follow acute than chronic sinusitis. Lack of constitutional resistance and extreme virulence of the infecting organism are put forward as reasons why one patient develops osteomyelitis when another, under precisely the same conditions, escapes.

This author divides osteomyelitis from nasal sinus suppuration into two classes: (1) the acute, with a duration of from three to twelve weeks; (2) the chronic, with a duration of from six months to two years. In spontaneous cases, the bone disease may be unsuspected until the sinus is opened surgically, but the appearance of an edematous swelling at some distance from the sinus should be regarded with suspicion at all times; a rise of temperature usually heralds the onset of this grave complication. In postoperative cases, there is little in the appearance of the parts to distinguish the case from one of simple reinfection of the wound area. But the slowness of development contrasts with the smart reaction that follows reinfection. The swelling around the open wound, however, is pale, puffy and edematous, merging imperceptibly into the surrounding tissue. If the wound is reopened, the bone will appear dead white or discolored, and if a piece be removed pus will ooze from the cut surface. Constitutional signs of toxemia will be in evidence. The abscesses are separated by seemingly healthy areas of tissue, "The disease burrows like a mole in the earth and, like a mole, throws up mounds here and there as it goes along." Surgical intervention may greatly modify the course; and marked remissions may occur which may lead to a false sense of security that the disease has been conquered. "Caution, therefore, is necessary and a considerable period—according to St. Clair Thompson, at least five months—should elapse before we release the patient from control."

Extradural abscess is practically invariable—being absent in only two cases, Claoue's (an antrum case) and the author's own; less commonly, we see thrombophlebitis of the intracranial venous sinuses, cerebral abscess and leptomeningitis. "The whole clinical picture is that of a low and chronic but

progressive septic infection, characterized by tardy and imperfect tissue reaction, attacking the bone of a region anatomically open to fatal secondary complications, and inducing toxemia and septicemia more often than pyemia. The periodicity of the symptoms which characterizes even the acute form of the disease, is highly suggestive of oscillating defensive powers, and thus tends to support the etiologic theory of a relatively heightened microbic virulence."

Once osteomyelitis has set in, the only chance of saving the patient lies in the immediate and entire removal of the diseased bone. The whole thickness of the bony cranium must be removed, down to the dura. Further, the superficial area of the bone resected must exceed the obvious limits of the disease. Intervention must be comprehensive; resection must be radical. In doubtful areas, it is safer to remove bone than to leave it, so that removal should be carried wide of the disease in all cases. The sacrifice of large sections of the bony cranium need arouse no apprehension, for if the case recovers the defect will be made good by osseous regeneration.

Wheresoever the disease has spread, there must the surgeon follow it, and its further advances must be forestalled and countered by the measures we have suggested. Failing these, there is little else to be done. Schilling has suggested, in desperate cases, the cutting of a linear gutter across the bony vault of the cranium from one temple to the other. Unfortunately this attempt to "ring" the disease, as woodmen ring a forest fire, would only be partial. The route to the vulnerable base of the skull would still lie open to attack. The use of vaccines and antitoxins suggests itself, but these remedies would be valueless if the diseased bone were not first removed.

H. B. Lemere[10] reproduces much of McKenzie's article, and adds eight cases reported since McKenzie's publication, together with one of his own in which extensive removal of bone and drainage failed to check the progress of the affection. X-ray pictures taken throughout the course of the disease are reproduced, indicating the progress of the osteomyelitis, which seems to have affected all the facial and cranial bones except the occipital. The primary cause of death was infection of the frontal sinuses; contributing causes, osteomyelitis of the frontal bone and basilar meningitis.

As stated in the beginning of this article, I have collected from American and European literature the reports of fifty-five cases of osteomyelitis of the frontal bone occurring as the result of or in connection with suppuration of the frontal sinus. To this list I have added, as taken from the literature, the reports of seven cases of osteomyelitis of the frontal bone due to suppuration of the antrum with extension by way of the frontal sinus to the frontal bone. In some instances the author has given a very meager description and few details, leaving considerable to speculation and perhaps some questioning as to etiology and findings.

I have attempted to present all of the case reports very briefly, in chronologic order, according to date of publication. They are as follows:

Case Reports.

1. Lannelongue[8]. Girl, age 14. Empyema of the left frontal sinus following measles, evidenced by swelling in the left frontotemporal region. Pus was liberated by incision of this abscess, and following this intervention there was severe headache, the pupils became unequal, and coma supervened, followed by death. Necropsy showed that necrosis of the anterior wall of the left frontal sinus had permitted the infection to extend to the diploë; there was a pus collection between the periosteum and the dura mater. The bone everywhere was infiltrated with pus.

2. Schilling credits Laurens with having observed this case in 1898, and Luc credits Laurens with having reported this case in Ann. de Mal. de l'Or., 1902, page 21. (This latter reference has not been verified as the article was not available.) Girl, age 10. Acute suppurative frontal sinusitis followed by subperiosteal and extradural abscesses extending beyond the superior longitudinal sinus. The posterior sinus wall, where the osteitis originated, was extirpated and a considerable area of frontal bone curetted, the dura being exposed within a circumference of fifteen cm. Regeneration of the bone after more than a year. Patient was exhibited four years after operation, being then in good health.

3. L. Müller[11]. Credited by Luc. Woman, age 23. Spontaneous osteomyelitis of the frontal bone and acute frontal sinusitis. Extension of subperiosteal abscess to the oppo-

site side. Though these were continually opened and drained, a "vast extradural abscess" eventually developed, and death resulted from leptomeningitis.

4. R. Botey[12]. Credited by Luc. Boy, age 16. Acute frontal sinusitis accompanied by osteomyelitis of the frontal bone, leading to infection of the dura mater and the underlying cerebral tissue. Improvement after the evacuation of the cerebral abscess was followed two months later by the second subperiosteal abscess near the vertex, with perforation near the parietal bone and a fatal meningitis.

5. Tilley[7]. Woman, age 22, almost complete bilateral nasal obstruction from polypi and granulations, associated with profuse discharge of pus from both nostrils and into the throat. Several subsequent operations required in an attempt to check an osteomyelitis which progressed as the author says "until the scalp was practically undermined." As a last resort, transverse incision was made completely across the scalp from ear to ear, beyond the edematous soft parts, in the hope of checking the spread of the disease, but the patient died after nine months' illness, most of the time having been free from pain. Postmortem examination showed necrosis of the vault extending a little below the level of the torcula; inner wall of the frontal bone healthy; dura mater intact throughout except that it was adherent all over the vault, which it practically held together; both sigmoid grooves were filled with pus, and about ten abscesses the size of hazelnuts were scattered through the cerebrum, some appearing as yellow patches upon the cortex.

6. Luc[13]. Man, age 20. Several operations for chronic purulent sinusitis covering a period of five years, the last followed by subcutaneous infection spreading rapidly over the forehead, during which the frontal bone was found extensively involved. Repeated curettements and treatment with strong solution of zinc chlorid failed to check the progress of the infection and the patient finally died from cerebral involvement. No autopsy permitted.*

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*The cases of Luc and Tilley were both reported at the 67th annual meeting of the Brit. Med. Assoc., and at that time, and in several articles since, they are credited as being the first recognition of the fact that osteomyelitis of the cranial bones may occur as a complication of nasal sinus suppuration. Though Luc's report appeared first upon the program, Tilley's case actually occurred a year before Luc's, so the Englishman should be accorded precedence in observa-

7. Roth[14]. (Recorded in Luc's article[2].) Man, age 39. Chronic frontal sinusitis. Following operation, a subperiosteal abscess formed in forehead. At second operation, extensive osteomyelitis discovered. Death from meningitis. Autopsy showed thrombosis of superior longitudinal sinus and abscess of the left frontal bone.

8. Lambert Lack[15]. Credited by Gerber. Two cases. Man, age 43. Acute inflammation of the bone fourteen days after operation on the right frontal sinus. The sinus wound was reopened and the area extensively curetted; a subperiosteal abscess was found in the frontal region; the infection rapidly extended and death from cerebral complications occurred seven weeks after the first operation. The infection had its origin in the anterior wall of the sinus.

9. Lambert Lack, second case. Girl, age 21. Bilateral frontal sinus operation. One sinus reopened on the fifteenth day, pus escaped, but the wound soon reclosed and gave no further trouble. Other sinus healed, but seven days after operation a puffy swelling appeared in the supraorbital region. Rise of temperature and severe headache. This abscess and others which subsequently formed were opened, and an attempt made to check the spread of the disease by making a transverse incision right across the frontal region, removing the whole external plate of the frontal bone, with curettage of the diploic tissue. Temporary improvement was followed by the extension of osteitis to the temporal and parietal bones; the mastoid became infected, and the patient died of cerebellar abscess.

10. Arnold Knapp[3]. Woman, age 21. Nasal occlusion and discharge for five years, and removal of at least fifty polypi from the nose with only temporary relief. Subperiosteal abscesses continued to appear after two external frontal sinus operations, and later an extradural abscess developed, after the evacuation of which the patient improved; but the

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tion. Following the publication of these proceedings, other case reports began to appear in the literature, sometimes several case reports by different observers appearing in one article, and oftentimes referring to early observations such as that of Laurens observed in 1898, but not reported until 1902. Therefore, in compiling the cases in chronologic order, the date of publication rather than the date of observation as claimed has been used, and the published record of cases as herewith compiled would seem to indicate that neither Tilley nor Luc are entitled to as much credit for observation as given them.

osteomyelitis continued to progress and death ocurred seventeen days after the last operation. Autopsy showed disease of the frontal bone "in a band shaped area one inch wide and extending from side to side. * * * No pus on the dura, and no evidence of the process having passed this membrane at any point." Commenting on this case, Knapp says that the morbid process began as osteomyelitis of the ethmoid bone, associated with empyema of all the accessory cavities of the nose; then an osteomyelitis of the frontal bone set in, extending to the squamous portion of the temporal bone on one side, and causing an epidural abscess with thrombosis of the sigmoid sinus, pyemia and death.

11. Schilling[4].* Reports four cases where osteomyelitis of the frontal bone followed frontal sinusitis.

(a) Woman, age 27. Seen by Schilling in April, 1901. Onset of bone condition was coincident with an acute right frontal sinusitis; an abscess formed beneath the periosteum over the posterior part of the sinus. Extensive removal of the osteomyelitic bone was followed eventually by recovery.

12. (b) Credited by Schilling as having been observed by Körner in December, 1901. Girl, age 13. Bilateral swelling over the frontal bones occurred during the course of an acute frontal sinusitis on both sides. On operation, osteomyelitis was found to extend upward as far as the hairline. Both sinuses were thoroughly curetted. A month later there was a sequestrum of the deep antral wall, with an extradural abscess. Following several operations and thorough curettement, the child eventually recovered.

(c) Laurens' case. See No. 2.

13. (d) Seen by Schilling in August, 1900. Girl, age 16. Had suffered from a chronic frontal sinusitis. Osteomyelitis followed operative intervention, and a series of subperiosteal abscesses were opened in succession, but the infection finally invaded the labyrinth and caused death from meningitis.

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*This is an extended and comprehensive discussion of the subject and the previously reported cases are analyzed, with suitable comments concerning their etiology, pathology and treatment. The history of the author's own two cases is given in great detail, with all the variations in his technic of treatment. The cases listed are of two kinds, first, those due to extension from the frontal sinus and, second, where extension was from middle ear. Frontal sinus cases include those of Körner, Laurens, Knapp and two by the author. It will be noted that the article contains no mention of cases of either Tilley or Luc.

14. Jacques[16]. Credited by Gerber. Man, age 63; bilateral frontal sinusitis; osteomyelitis of the anterior wall on one side; spontaneous symmetric dehiscence of the posterior walls. There had been a nasal discharge for six years, and six months before an abscess in the eyebrow had been opened. Pus and polyps in the nose. Some headache. Operation disclosed bilateral osteomyelitis of the anterior wall of the right frontal sinus, and on each side a defect in the posterior wall as large as a franc piece. The dura mater on the right was infected and granulating, but healthy on the left. Eventually, healing and recovery.

15. Durand[17]. Credited by Luc. Girl, age 18. Nasal obstruction and suppuration for many years, with several operations, following the last of which an edema appeared at root of nose, with development of a fistula through which denuded and softened bone was found. Operative intervention disclosed an osteomyelitis with four sequestra, and the dura was found infiltrated and suppurating. Death from intracranial complications.

16. M. Paunz[18]. Credited by Luc. Boy, age 17. Operation disclosed softened bone and fistula in the diploë toward the bottom of the superior wall of the sinus. Despite two operations, the patient died from leptomeningitis. The author believes that the intraosseous vein, the course of which could be traced through the friable bone tissue which surrounded it, was the avenue of infection from the frontal sinus to the endocranium.

17. Capart[19]. Credited by Gerber. Male, age not given. Staphylococcus infection of both frontal sinuses communicated to the cranial bones and discovered in the course of bilateral radical operation. Death.

18. Winckler[20]. Credited by Gerber. Woman, age not given. Acute frontal sinusitis for three days, when operation was undertaken and osteomyelitis was found involving the bony walls of both frontal sinuses, including the lower walls of the orbit. Patient eventually recovered.

19. Röpke[9]. In this article, the author has something to say concerning priority of cases. He reviews the literature, and states that up to the period at which he was writing, but thirteen true cases of frontal bone osteomyelitis originating in the frontal sinus had been reported. To these he added three

of his own. (a) Woman, age 23. Operated four years previously for an acute bilateral frontal sinusitis, which apparently had been cured. A recurrence required a second operation, and later intervention was required on account of the development of extensive osteomyelitis and a complicating extradural abscess. Partial relief from the operation, and a month later revealed a huge extradural abscess. Death from intracranial complication.

20. (b) Man, age 23. Suffered from chronic suppuration of both frontal sinuses; and following operative intervention, osteomyelitis set in on left side of forehead, extending rapidly to the hairline and complicated by an extradural abscess. An extensive resection of the bone was carried out, but death occurred from the intracranial complication.

21. (c) Man, age 26. Chronic frontal sinusitis, which upon operation disclosed necrosis of the anterior wall of the frontal sinus and the bone infiltrated with pus. Extensive curettement, followed later by the appearance of sequestrum. Patient finally recovered.

22. Keimer[21]. Credited by Luc. Girl, age 13, without previous nasal trouble. An edematous swelling appeared on the right side of the frontoorbital region, accompanied by fever. Sinuses opened, curetted, dura found intact. Subsequently, subperiosteal abscesses formed on the parietal and later upon the occipital bones. Each time the affected area was widely curetted. Improvement at first, later clonic spasms on the left side followed by temporary paralysis, but eventually recovery.

23. Killian[22]. Two cases. No sex or age given in either case. Both credited by Luc. In the first case, there was rapid necrosis of practically the entire frontal bone in the course of a chronic frontal sinusitis, and producing a fatal meningitis. No details given.

24. In the second case, there had been a pansinusitis ten years before. Osteomyelitis set in following the performance by Killian of his operation. Four successive resections failed to stop the progress of the disease. The infection first gained entrance to the temporal bone, then the temporosphenoidal lobe by way of the dura mater, where an abscess developed which caused death of the patient by opening into the ventricle.

25. L. A. Coffin[23]. Credited by Luc. Woman, pregnant three months, age 33. A subperiosteal abscess developed during the course of an acute frontal sinusitis. Abortion took place, and the skull was opened in an attempt to drain a brain abscess, which however proved fatal. In commenting on this case, Coffin says he thinks that the infective agent had entered the bone and osteomyelitis and pyemia already were established when the patient was seen first.

26. Swain[24] (reported from memory). Credited by Gerber. Sex and age not given. Chronic frontal sinusitis. Operation followed by osteomyelitis and death from intracranial complications.

27. Luc[2]. The author reports a second case, the first (see Case 6) having been reported at the Brit. Med. Assoc. and recorded in the transactions of that organization. Man, age 30. Operated for acute right frontal sinusitis, and the infection was found to have extended to the bone, forming an extradural focus. The process extended, and during a period of two years, numerous operations were performed to check extension of the osteomyelitic process, which, to quote the author, extended "over a vast surface" and required extensive curettement with exposure of considerable spreading dura mater. Patient eventually recovered.

28. Mouret[25]. (Unpublished communication) November, 1902, recorded in Luc's article. Man, age 29. Spontaneous appearance of osteomyelitis with acute frontal sinusitis. A subperiosteal abscess formed some distance away. Death took place by intracranial infection.

29. Bertemas[26]. (Unpublished communication but no date given for this case.) Credited in Luc's article. Girl, age 9. Osteomyelitis in connection with a bilateral hyperacute frontal sinusitis. On operation the suppuration was found to communicate with an extradural abscess. Extensive resection, but death followed from the intracranial complication.

30. Schmiegelow[27] (Sept.-Dec. 1900, unpublished communication). Credited by Luc. Man, age 39. Following an incomplete operation on the right frontal sinus for chronic suppuration, osteomyelitis set in, which extended to the temporal region. Despite wide resection and curettage, the man died of meningitis. The greater part of the frontal bone was necrosed.

31. Macewen-Miller[28]. Credited by Luc. Young girl, age not given. After operation for chronic frontal sinusitis, pyemia developed with subperiosteal abscesses in the frontal region. Death from pulmonary and intracranial complications.

32. P. H. Gerber[1]. Girl, age 17. Chronic frontal sinus suppuration, followed by pansinusitis. Twelve months of conservative treatment failed to cure. Operation disclosed hemorrhagic areas in the bone and a circumscribed bluish discoloration. Complete removal of the diseased bone and resulting ultimate regeneration and recovery.

33. Forestier[29] (personal communication). Credited by Gerber. Male, age 48. Subacute primary frontal sinusitis, osteomyelitis of the anterior wall. Coryza two months before. Coma, perforation of the anterior wall of the sinus. Operation showed invasion of the dura mater, the diploë having transmitted the infection from the sinus to the brain. Death from septic meningoencephalitis.

34. Morel and Hubert[30]. (Date and reference not given.) Credited by Gerber. Man, age not given. Operation for frontal sinusitis disclosed osteomyelitis with sequestrum in the right temporal fossa. Operation failed to check spread of infection, and patient died from meningitis twenty days later. Autopsy showed osteomyelitis of the frontal, temporal and parietal bones.

35. Logan Turner[31]. (Personal communication.) Credited by McKenzie. Male, age 30. Two radical operations by different operators for chronic frontal sinusitis, followed by development of fistula at root of nose, and some edema of soft parts. Practically all of the frontal bone removed at successive operations for the osteomyelitis. Death from pyemia, which definitely spread from the frontal sinus. Never any definite signs of meningitis or brain abscess.

36. Van den Wildenberg[32] (Antwerp). Credited by McKenzie. Female, age 23. Endonasal operation on an acute bilateral frontoethmoidal sinusitis. Recurrence treated by radical operation on left side. Three weeks later, osteomyelitis on right side, with appearance of sequestrum. Succeeding operations failed to check progress, and patient died from intracranial complications. Osteomyelitis was thought to have originated in the wall of the right or nonoperated frontal sinus.

37. Von Eicken[33]. Two cases. Sex, age and dates not given. Case one. Credited by McKenzie. Chronic frontal sinus and ethmoidal suppuration with acute symptoms after accidental traumatism. Operation disclosed osteomyelitis which repeated operation failed to check. Death from meningitis.

38. Second case of Von Eicken. Osteomyelitis of frontal bone followed by a radical operation and finally resulting in intracranial complications and death.

39. Wolff Freudenthal[34]. (Date and reference not given.) Credited by McKenzie. Male, age 25. Acute frontal sinusitis during which osteomyelitis developed and was discovered at operation. Extradural abscess formed and evacuated, but no relief. Later, brain abscess located and drained, with recovery.

40. Charters Symonds[35]. Credited by McKenzie. Male. age 30. Chronic frontal sinusitis. Osteomyelitis discovered at operation. Extensive removal of frontal, part of sphenoid, roof and inner wall of right orbit and orbital ledge. Eventual recovery, and the greater part of the frontal bone was reconstructed, with the consequent restoration of the elevation of the forehead.

41. E. D. Davies[36]. (Personal communication) 1908. Credited by McKenzie. Female, age 20. Chronic frontal sinusitis, with several operations, the last, a radical, being followed by osteomyelitis of the frontal bone requiring extensive curettement. No effect from staphylococcus vaccine. Patient fully recovered.

42. Walker Downie[37]. Credited by McKenzie. Male, age 36. Chronic frontal sinusitis, with development of osteomyelitis involving the outer table of the frontal bone. Operation and recovery of patient.

43. McKenzie[5]. Male, age 28. Several operations, including a radical for chronic sinusitis, followed by osteomyelitis requiring repeated curettements. Autogenous vaccine administered. Death from meningitis. The autopsy indicated that the infection reached the meninges by extension of suppuration back under the roof of the orbit.

44. W. G. Porter[38]. Credited by Lemere. Woman, age 46. Developed frontal osteomyelitis six months after operation on the frontal and other sinuses, and reovery followed after extensive removal of the diseased bone.

45. Mollinson[39]. Credited by Lemere. Boy, age 15. Extensive osteomyelitis discovered during frontal sinus opera-

tion. Patient recovered following wide removal of diseased bone.

46. Normal Patterson[40]. (Reported at same time Mollinson's case was reported and credited Lemere.) Male, age not given. Osteomyelitis followed a frontal sinus operation, and after twelve operations were performed patient finally recovered.

47. H. L. Lynah[41]. Credited by Lemere. Female, age 13. Simultaneous development of frontal sinus suppuration and osteomyelitis of the frontal bone as a complication of scarlet fever. Recovery followed removal of squestra and softened bone from frontal, temporal and malar bones. (Pus still was accumulating when case was reported.)

48. Herbert Tilley[42]. Report of a second case. First already noted. Credited by Lemere. Woman, age 35. Six weeks after operation for frontal sinusitis, was operated again for osteomyelitis, and the whole left frontal bone together with inflamed dura and brain tissue was removed. Sequestra appeared for several months but patient eventually recovered.

49. Ralph Opdyke[43]. Credited by Lemere. Boy, age 18. Had left frontal sinus curetted, and eleven days later a trephine operation, at which seropurulent fluid was aspirated from the fourth ventricle. Ten days later, removal of parts of the parietal, occipital and temporal bones. Several weeks later, a double sinus operation. After twenty-one operations the patient recovered.

50. G. B. Wood[44]. Two cases. Credited by Lemere. First case: Male, age 15. Operation for acute sinusitis followed by the development of osteomyelitis. Operation failed to check progress of the process, and patient died from intracranial complications two months after the onset.

51. Case two: Male, age 24. Developed osteomyelitis after a frontal sinus operation, and numerous abscesses were opened and diseased bone removed; but trephining was necessary later "for a brain complication," and the patient finally died after four months.

52. J. H. Bryan. Credited by Lemere. Male, age 15. Spontaneous frontal sinus suppuration and localized osteomyelitis. Operation failed to check spread of the process, and death resulted eleven months later from intracranial complications.

53. H. B. Lemere[10]. Male, age 32. Acute frontal sinusitis, which on operation showed perforation of the upper wall

of the orbit and epidural abscess. Extensive removal of bone. Thirteen abscesses of frontal bone, lids and cheek were opened during a period of two months, each time with evacuation of large quantities of pus. Intracranial complications caused death of the patient three months after first operation. Progress of the osteomyelitis recorded by X-ray pictures. All the cranial and facial bones except the occipital were affected. The author says the primary cause of death was infection of the frontal sinuses, and contributing causes were osteomyelitis of the frontal bone and basilar meningitis.

54. R. Lund[46]. Male, age 38. At operation for an acute frontal sinusitis, a subperiosteal abscess was discovered and the orbital wall was opened and curetted. A month later, a single operation for extensive osteomyelitis of frontal bone. Process progressed, and eventually meningitis developed with death of patient.

55. C. F. Warren[47]. Male, age 19. Operation for acute frontal sinusitis was followed twenty-one days thereafter by osteomyelitis requiring removal of large portion of frontal bone. The scalp flaps were sutured back and warm eusol applied every four hours. Healing progressed satisfactorily, and patient was discharged in four weeks. Author attributes success to the very free removal of bone, the open method of dressing, and the continuous application of eusol to the wound.

Antrum Cases.

1. St. Clair Thomson[48]. Credited by Gerber. Female, age 27. Chronic bilateral sinusitis, the infection apparently beginning in the maxillary antrum. The author reports that the osteomyelitis of the frontal bones occurred through extension by way of the wall of the frontal sinus. Three operations failed to check the progress of the osteomyelitis, and the patient finally died from intracranial complications.

2. Claoué[49]. Credited by Gerber. Woman, age 44. Maxillary sinus operation fourteen years before; empyema of frontal and maxillary sinuses, edema and abscesses of lower lid and root of nose. Four operations were performed, the third being curettage of the frontal bone on both sides; lumbar puncture; epileptiform seizures; death. The course of the infection was through the anterior wall of the right frontal sinus to the diploë and the superior longitudinal sinus. Autopsy showed

intradural abscess, thrombosis of the longitudinal sinus and abscess in the motor zone.

3. H. Wokenius[50]. Male, age 16. Chronic maxillary sinusitis, which eventually was communicated to the ethmoid and frontal sinus on the left side; and later produced a streptococcic osteomyelitis of the frontal bone, which the author describes as a fulminating osteomyelitis, resulting in death four days later. In commenting, the author says the notable feature of the case is the rapid and stormy course of the disease, and he believes that no other case of such brief duration has been recorded.

4. Neil McClay[51]. Credited by Lemere. Man, age 30. Ostomyelitis of frontal bone set in two months after operation upon antrums, ethmoids and frontonasal duct, and it was necessary to remove the whole anterior wall of the frontal sinus and a large portion of the table of the left frontal bone. Later, most of the left and part of the right frontal bones were removed. The patient finally recovered.

5. Ross H. Skillern[52]. Credited by Lemere. Male, age 28. One month following the removal of spur of the septum, patient had operation on the maxillary antrum, and still nine days later had opening of the frontal sinus with evacuation of pus. Still later, when Skillern first saw him, the maxillary sinus was reopened and much softened bone removed, and ten days thereafter the alveolar process came away in two large sequestra. Still later, both frontal sinuses were found involved, as well as the orbital wall of the right frontal sinus. Numerous radical operations and removal of the osteomyelitic bone failed to check the process of the disease, and the patient finally died from general septicemia.

6. Herbert Tilley[53]. Male, age 45. Right sided pansinusitis with profuse discharge of pus. Ordinary Caldwell-Luc operation performed, followed within five weeks by an operation that disclosed osteomyelitis of the frontal bone which even repeated operations failed to check, and within fifteen weeks the patient died from intracranial complications. The author says that this is the first case in his experience where an operation on the antrum lit up an acute spreading osteomyelitis of the maxillary and frontal bones. He believes that the bone inflammation started in the antrum, because the initial symptoms were limited to the cheek and the lower eyelid and the surrounding soft tissue.

7. Herbert Tilley[53]. Male, age 60. Two operations on the antrum had been performed. Irrigation of the right frontal sinus caused the soft tissue of the forehead to bulge, indicating that the anterior wall was more or less destroyed. Three weeks later, an operation revealed the anterior walls of the frontal sinuses necrosed, infiltrated with pus, and so soft that they could be removed with a blunt curette. Several subsequent operations for the removal of the osteoymelitic process were unavailing, and the patient eventually died from intracranial complications.

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DISCUSSION.

DR. ROSS H. SKILLERN, Philadelphia: Osteomyelitis of the frontal bones is such a complicated and rare condition, that it offers many points which could be discussed with interest and profit. However, I propose to touch on only two or three of these, which in my opinion are of paramount importance.

In the first place, the etiology. Syphilis is one of the suspicious etiologic factors in this disease. I recall two cases I have had in which I failed miserably because I trusted too much to what the patient told me, even though I was suspicious of lues.

Trauma plays an important role in this as well as mucocele, and also in the chronic variety of frontal sinuitis. And in osteomyelitis, we must remember the peculiar susceptibility of the individual to the germ at that particular time. Two people are sitting together, perhaps in a theater; one takes cold and the other does not. It is because one of them is particularly susceptible at that moment. I think that is exactly what occurs in these cases of osteomyelitis—the patient is particularly susceptible to the infecting organism at that particular time.

As to treatment, the cases that have their inception in an operation are hopeless, so far as I have been able to learn—all die, despite the most drastic measures to stop the progress of the infection. The English sur-

geons even went so far as to take a Killian chisel and make a linear gutter across the vertex, through the parietal bones, from ear to ear, but in vain, as the disease progressed to the other side by way of the blood stream. The spontaneous cases, apparently, are the only ones that give any hope, and these depend upon the type of infection. It must be a pure culture of staphylococcus aureus. The streptococcus spreads like wildfire, with death in a short time. The spread of the pneumococcus is slower but ruthless, and in the end just as sure. However, if we have only the staphylococcus aureus to deal with, we should persevere until the very end, for success may follow when hope has been abandoned, as has been shown by the many cases of recovery cited in the literature on the subject.

DR. H. B. LEMERE, Omaha, Neb.: Dr. Bulson's case is unique, in that it resulted from such a slight trauma. It is partial evidence, at least, that excessive trauma at time of operation in frontal sinus cases cannot be held accountable for progressive osteomyelitis. The reason that operated cases of frontal sinusitis are more fatal than spontaneous cases is somewhat obscure. It may simply be that operated cases are generally those which show such pronounced and long standing infection, that they, as a class, are more profoundly affected systemically and locally than are the spontaneous cases, and it is because of these urgent symptoms that operation is considered necessary and often imperative in order to prevent involvement of the brain or meninges. Therefore, the fact that operative cases are often fatal should not be considered an argument against external operation on the frontal sinus in cases in which it is indicated. I reported a case in the Journal of the A. M. A., March 3, 1923, Vol. 80, pp. 596-599, and in this report I brought, as far as I was able, the list of the reported cases up to that date. In this case, complication of the brain and meninges was imminent, and the case was an emergency operation. Many more cases are reported in the English literature than in our own, either because the condition is more prevalent in England, or because we fail to report our cases in the literature. Dr. Bulson's case is valuable as adding another recovery to the list of those in which there was an extensive removal of bone, and this I think should be the procedure as soon as we are sure from the progress of the disease and from our X-ray findings that we are dealing with a progressive osteomyelitis.

I wish to report another case of progressive osteomyelitis involving the lower part of the head and resulting in erosion and rupture of the internal carotid: Lillian Kinder, age 29, single, fairly well nourished, somewhat pale. Previous history uneventful. Childhood diseases—measles, mumps, pertussis, chicken pox, small pox. Appendectomy five years ago. Tonsillectomy three years ago. Complains of sinking spells and shortness of breath occasionally. One year ago, had attack of acute indigestion. Since then has had bad headaches in the left parietal and occipital regions and foul discharge from the left nose.

EXAMINATION: There is foul odor from left nose. The middle turbinate is denuded of mucous membrane, and stony calcareous bone presents. The pharyngoscope shows inflamed adenoids and inflammation around the Eustachian tube, but no further detail in the nose. Tonsils have been removed clean. X-ray shows right antrum slightly cloudy(?); left antrum + + +; right frontal and ethmoid(?). The patient's complexion was sallow, her heart action weak and she appeared sick. Temperature normal.

There were no stigmata of syphilis and Wassermann was negative. A diagnosis was made of probable osteomyelitis, and a grave prognosis given.

OPERATION: Under local anesthesia, an attempt was made to clear out the necrotic tissue. Owing to pain, this operation was completed under ether and through and through drainage made into the antrum. The mucous membrane of the left antrum was pyogenic and velvety. There was no retained pus. Healing was uneventful and the odor disappeared from the nose with only slight discharge persisting. The patient went home.

June 9th, 1924. The patient returned feeling badly. Nose on left side blocked with granulation. June 10th, 1924. Operation. Partly under local and partly under general anesthesia, the left antrum was opened and a large amount of granulation tissue removed. The ethmoid was carefully exenterated. After the operation, clear fluid exuded from the nose at the rate of two drops per minute. This was collected and submitted to the serologist, who reported that it did not reduce Fehling's solution and was not cerebrospinal fluid. The technic of the operation was such, that assurance was felt that the floor of the skull had not been injured. The clear discharge continued for four days and then stopped. At no time were there meningeal symptoms, and the autopsy later showed no injury or pathology of the meninges or floor of the anterior fossa, this region being carefully examined. The sphenoid and frontal were, however, filled with straw colored clear fluid, which was probably identical with the discharge from the left nostril. A swelling appeared over the left hard palate, and later a circular area of necrosis one inch in diameter occurred, involving the hard palate and posterior palatal process of the superior maxillary bone. The patient continued afebrile till a few days before her death, which occurred July 13th, 1924, from rupture of the internal carotid into the nasopharynx. Calcium lactat, cod liver oil, thyroid extract, urotropin, and the day previous to death intrevenous mercurochrome, were all used. The cod liver oil seemed the most beneficial, but failed to check the progress of the osteomyelitis.

During the whole course of the disease there was extreme sensitiveness of the left nose to touch. This was only partly controlled by cocain. There was also severe left headache and pain on the left side. The autopsy findings of the extension of the osteomyelitis to the bone lying next to the Gasserian ganglion seems to account for this.

Death in these cases usually results from invasion of the cranial cavity along the sheaths of one of the cranial nerves, but this result was anticipated in this case by the rupture of the carotid. This case is reported briefly as an addition to the case of osteomyelitis of the frontal bone previously reported by me, in which report there was made a summary of the literature to date. (See above.)

AUTOPSY: Dr. J. J. Keegan, Department of Pathology, University of Nebraska, Medical College, Omaha, Nebraska: The body was that of a well nourished young woman. Examination of the mouth showed a darkened area with a necrotic center, from which yellow pus exuded over the junction of soft and hard palate. The soft palate was removed and showed a rough area about 2 cm. in diameter, dark in color on the posterior surface. An area of dark sloughing tissue was found in the pharyngeal recess, high in the left side. The skull cap was removed, showing a very thick skull. The dura was smooth and not adherent. The brain was removed

and found to be apparently normal. The dura was stripped from the base of the skull. An area of necrosis in the petrous portion of the temporal bone was found below the similunar ganglion on the left side. The cavernous sinus on the same side was opened, the internal carotid artery picked up and traced downward; clotted blood was found, and in the carotid canal, a point in the anterior part of the artery was found which had eroded away. A probe was found to pass from this point into the pharynx through the necrotic area in the pharyngeal recess described above. The left mastoid cell contained a straw colored mucous fluid. The right mastoid cells were clear. Neither middle ear showed any pathology.

DIAGNOSIS: Osteomyelitis of the superior maxillary, sphenoid, and temporal bones; erosion of the internal carotid artery in the carotid canal.

DR. JOHN J. SHEA, Memphis, Tenn.: I want to report two cases and will show slides.

Slide 1. This is a boy seen several weeks after he had had a double radical frontal operation done in a neighboring town. He came to us with a wound over the site of the fontanelle, with an osteomyelitis, as you can see, involving an area extending for about three square inches. At operation, it was necessary to remove at least four square inches of the outer table, and the inner table was lost for an area of the size of a half dollar over the site of the fontanelle.

Slide 2. This was another lad with practically the same loss of tissue, but not quite so extensive. He had lost the outer table and part of the inner table, both at the site of the fontanelle. Both of these cases were adolescents, one fourteen and the other twelve. This lad developed an extradural abscess, and there is a tube into the abscess. He is now 18 months past the operation and well. The other lad is now in the hospital, still in the acute stage.

DR. J. M. WAUGH, Cleveland, Ohio: Within the last year or two, several interesting reports have been made of osteomyelitis of the skull. I will show, in a moment, several slides of a very extensive osteomyelitis which occurred in a case seen at the Clinic this past spring.

Three or four years ago, this woman, about 40 years of age, had been sent to Colorado with a diagnosis of tuberculosis. She had improved rapidly and, so far as she knew, her tuberculous condition had been healed for a matter of two years. She was quite well until Thanksgiving day of 1924. On that day, she rose with a severe headache accompanied by a cold, which continued to increase. Two or three days later, she saw a specialist, who very shortly afterward did a rather incomplete radical frontal sinus operation. I say incomplete, because there was apparently no drainage from the sinus itself down through the nose, and according to the patient's history there had been none. There had never been complete closure of the external wound, and a discharging sinus existed at the time of her examination. She had developed headache, at first intermittent then more or less continuous, was markedly septic and had been taking a good many drugs for the control of the pain, so that her digestive disturbance was very marked and her prostration was profound.

The X-rays, as you will see from the plates, showed a very extensive osteomyelitis of the entire frontal bone and some involvement of the parietal bone. Because of her previous history of tuberculosis, I was anxious to exclude this as a factor, and from all our findings, there was no tuber-

culous element in the present examination. She had an old healed tuberculosis in her chest, and to confirm the above opinion, under local anesthesia, I removed a small area from the external plate of the skull, which was sent to the pathologist and reported as a chronic inflammatory osteomyelitis with no evidence of tuberculosis. Lues was excluded both by blood and by spinal fluid tests and there was no history about the patient that would suggest lues.

She left the hospital in a very much weakened condition, at the insistence of her relatives, and was taken to another part of the state. I did not think any operation, no matter how radical, would assist in this woman's recovery.

It is well to remember that the pain in these cases is often intermittent, which, I think, is due to the involvement of new areas of periosteum. If the process is not very active, the patients do not have an extreme amount of pain, but you are impressed with the profound weakness and evidence of absorption which depresses the patient tremendously.

I believe the records reported in most cases show that extensive osteomyelitis of the frontal bone usually follows radical surgery on the frontal area.

Dr. Thomas E. Carmody, Denver, Colo.: I have seen two cases of this kind. The first one had a positive Wassermann and was a frontal sinus case. However, we considered, after the operation of this case, that the osteomyelitis was not due entirely to the infection, but probably to three doses of salvarsan that were administered. We felt the injury and sudden breaking down of the bone was probably more from the arsenic than from the infection, although the infection was an important point, and he had had a chronic frontal sinuitis for years. This case did not have an exposure of the dura, as I remember. The outer table was necrotic clear to the coronary and over both frontal sinuses. He recovered after two or three operations at my hands, and then he selected another surgeon under whom he recovered.

I had the second case this Summer, a case of perhaps three years' duration, a pansinuitis. He had chronic frontal and chronic maxillary sinuitis. He had a fistula into the left orbit which had been present for three years. At the first operaton, a radical frontal was performed, with window resection of the maxillary. A week later it was necessary to open the frontal because of a periosteitis, and because of edema and pus formation over the maxillary sinuses, to open the maxilla and open externally on the cheek. About three weeks later, I opened up the frontal in the median line, clear back to the corona, and evacuated a large amount of pus, with a great amount of exposure of the inner table and perforation in the median line into the dura. This patient died within one week after the last operation of meningitis.

Dr. John McCoy, New York City: These cases are not as infrequent as Dr. Bulson would lead us to expect. I have had three which I have not reported as such. One was due to the fact that the patient was treated by an eye specialist for orbital abscess in both eyes. I saw him in consultation six or seven weeks afterwards, made a broad incision on both sides and evacuated pus, and on the left side I found an osteomyelitis extending to the vertex of the skull. That was a child three years of age. The child left the hospital in ten days, came to my office for treatment

for about two weeks and was apparently doing well, but at the end of that time there occurred a slight paralysis of the opposite side. The child was seen by a nerve specialist, who said that perhaps the bandage was pressing on the motor area. After a few days, I was called and found the child unconscious. On opening the frontal lobe, we got out a cupful of pus. The child unfortunately passed away.

The second case I saw in consultation, about five weeks after an incomplete frontal sinus operation. This was a young boy, about twelve, and when I saw him, the principal complaint was a swelling on the forehead at the hair line. Suspecting there was a connection between this and the operation, because there had apparently been imperfect drainage, we opened the whole area and were surprised to find that the posterior wall of the frontal sinus was necrotic, there were granulations on the dura, coming out from here as a swelling at the hair line. This boy progressed fairly well, but had periods of somnolence. These becoming greater, we decided to open the frontal lobe, which we did and found a large abscess. For about 48 hours he apparently did well, but then commenced to have temperature and somnolence again. Then we tried a procedure that worked very successfully. We made counter drainage in his temple, making drainage through and through from the frontal wound to the temporal wound. This case resulted in a cure.

The third case was one in which I was not sure of the etiology, because the man had a chronic frontal sinuitis and developed subsequently a swelling on the top of the skull. Having in mind the previous case, we opened this area and found an osteomyelitis, apparently of the parietal bones. We extended our incision as far forward as the bone was diseased, but we never were able to connect his osteomyelitis with the sinus trouble, so whether it was a metastatic affair or not I do not know. He did not heal, however, after the first or second incision, but did after the third incision. I think this case took about seven or eight months. Subsequently, a very beautiful operation was done by Dr. Joseph E. J. King, who made osteoplastic flaps over the skull and covered over this defect. I have not reported this case.

DR. H. P. MOSHER, Boston: This paper is painfully interesting to me, because I have in the hospital now a case that I am not particularly responsible for—it is a charity case done by one of my associates, a radical frontal operation following osteomyelitis. I am worried because Dr. Skillern says no osteomyelitis of the frontal bone ever recovered. Most of the cases of osteomyelitis I have had have been cases following operation on the temporal bone, but we have had in the hospital three cases of osteomyelitis of the frontal, including the one I just mentioned.

A word as to the eitology, and then finally as to the operative procedure. I have a feeling, and our X-ray man has the feeling, that the extension of the infection is often by the antrum. Two of these cases of osteomyelitis came after the extraction of teeth, and if you investigate the antrum following the extraction of teeth, you will often find the seat of the trouble.

About the operation: I do not like to be called conservative, because that is another way of spelling a word of three letters which a gray haired man does not like. In the beginning, I went at these cases hammer and tongs. Dr. Bulson has an assurance as to the operative procedure in these cases which I do not share. I have given up maximum surgery in these

cases, and the reason is the lesson I learned from a case carried through by one of the younger doctors. This was an osteomyelitis involving the ethmoid and frontal. This man started in with enthusiasm, but every time he operated he spread the osteomyelitis. Then he turned around and said "I am spreading this: I will wait." He waited and used heliotherapy and cod liver oil and all the old-fashioned remedies, and finally, after waiting four months, the case is now cured. So I think there is such a thing as attacking these cases too radically. Dr. Beck said something yesterday about waiting before you opened a mastoid, in other words, before you go down and tie the jugular. He forgot that there was a time in his career when he opened mastoids very early—within the first week. But he was led to change his mind, and has changed it evidently, because now he says there is such a thing as going too fast.

I want to sound a warning about these operations that are so spectacular, and I want to raise the question whether it is not worth while to stand by with a little more judgment and less courage in the treatment of these cases.

I would like to know how many of these follow radical operation on the frontal, where there is a window opening in the anterior wall of the frontal.

Dr. George W. Boot, Chicago: I think the speakers have been confusing two different conditions. It is about twenty years since I read—an original observation as I remember—of a rapidly spreading osteomyelitis, which spreads through the venous channels so that the whole cranium is involved. I have been looking for this condition ever since, but have not found it.

The other condition is a localized osteomyelitis, which is common, and I think Dr. Skillern is right in what he says. I recall one case, a negro of about 35 years of age, who had a chronc sinuitis with a foul smelling discharge from the nose, and a fistulous opening in the forehead, but no other very serious symptoms. I operated on him and found a sequestrum one inch wide and two inches long. The dura was exposed, and we found an accumulation of pus. The man got well without any other complications.

Dr. Albert E. Bulson, Jr., Fort Wayne, Ind., (closing): Some difficulty is encountered in analyzing and classifying cases properly. Several of the cases of osteomyelitis of the frontal bone reported in the literature are open to question, as has been pointed out by some of the authorities quoted.

We are discussing, as Dr. Boot says, a rapidly spreading destructive inflammation of the frontal bone, and it is a serious condition, as evidenced by the reported fatal cases in the care of some of the most prominent and experienced operators in this country and abroad. That many cases have not been reported is undoubtedly true. Our confreres should report these cases, and when they do so the reports should include roentgenograms, bacteriologic findings, blood examination, and a full record of each case.

The character of the infection has a good deal to do with the progress of the disease. As Dr. Skillern has pointed out, those cases that are most typical are due to staphylococcus and pneumococcus infection. The

spreading and destructiveness of the inflammation depends upon a lowered resistance and the virulence of the infection.

I agree with what Dr. Mosher said concerning conservative treatment, and the advisability of avoiding radical and mutilating operations when possible. However, the history of all of these cases shows that we are dealing with a destructive process that is not self limited and does not show any marked tendency to get well under anything but radical measures. Therefore, I can not agree with Dr. Mosher in the statement that conservatism in such a virulent and rapidly progressing trouble is the wise thing, especially when we know from the experience of many operators that the trouble often ends fatally. I believe that the operative cases to which Dr. Mosher has referred would have gone to the bad anyway, and that death occurred in spite of the operative interference and not because of it.

Twenty years ago, I saw in consultation what I now believe to have been two cases of osteomyelitis of the frontal bone, in the hands of a first-class general surgeon. I remember hearing the surgeon say that he believed Nature had a tendency to wall off this infection, and that laying open the sinus for drainage was quite sufficient. I wanted him to remove a good portion of the diseased bone, but he refused. The destructive process spread rapidly and death ended the career of the patient. The second case was very similar, and I believe if it had received some radical operative attention, the patient might have gotten well. I have not reported these cases, as I did not have enough details to justify it.

The syphilitic cases have been eliminated entirely, as I do not think they belong in this class of cases at all. McKenzie, also, has eliminated syphilitic cases entirely.

I believe that operative interference in these cases is not only justified, but that it should be carried to the point of radicalism, and I am glad to have had the opportunity of presenting this case which recovered following several operative interventions. I feel quite certain that the literature of Europe as well as America has been scanned very thoroughly to get all of the cases reported, which number fifty-five all told. Since I have been here, several surgeons have informed me that they have had cases. I wish they would report their cases and give all the details.

TRANSACTIONS

OF THE

THIRTIETH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OPHTHALMOLOGIC DIVISION

CASE REPORT OF BILATERAL GLIOMA WITH ATROPHY OF ONE GLOBE.

B. S. Guyton, M.D., Oph. D., F.A.C.S.

OXFORD, MISS.

R. N. B. Male, age 8 months. On November 17th, 1924, at eight months of age, this child was brought to my office for treatment of a very acute inflammation of the left eye. The history up to this time was negative, with the exception of some digestive disturbances earlier in life. It had not grown well until after the fifth month. Although the prophylactic silver nitrat had not been used in its eyes at birth, there had been no eye trouble until this time.

The first impression on observing the eye was of gonorrheal ophthalmia, but the absence of pus was noticeable. The lids were so swollen it was with difficulty the eye could be opened. The conjunctiva was folded and protruding out between the lids. Ophthalmoscopic examination was almost impossible. Although the discharge was very slight, a smear was made for examination.

Urinalysis was negative. Family history was negative for lues and tuberculosis. Blood Wassermann for both father and mother proved negative. A general examination by an internist gave negative findings. Mercurochrome and ice compresses were prescribed.

Three days later, the child came back greatly improved. The smear had not revealed a Neisser infection, and at this time a zinc sulphat prescription was substituted for the mercurochrome, and atropin prescribed.

Five days later, when the child was seen again, for the first time a satisfactory examination of the globe could be made. Iritis was evident, with posterior synechia. A yellow reflex from the posterior chamber was noticeable, which, in view of the acute inflammatory course the eye had just gone through, was thought to be an exudative condition. No blood vessels could be observed. Very quickly all inflammation subsided, and the globe was seen to be shrinking quite fast.

The patient was not seen again until January 8th, about seven weeks after the first visit to my office and five weeks since the last. The mother was much distressed over the pupil of the right eye having the same yellow reflex that had been noticed in the left eye. It was at this time that she first told me of having noticed this reflex in the left eye two months before having come to me for the acute inflammation. Nothing wrong had ever been noticed in connection with the right eye until two days previous to this visit.

On examination, the left eye, the one originally affected, was found to have very little if any inflammation, but had shrunken greatly. At this time it was probably not more than one-fourth the original size, and apparently totally blind. The right eye showed distinctly the typical "amaurotic cat's eye." It had no inflammatory signs or symptoms corresponding to the course the other eye had run.

The pupil was slightly dilated but reacted to light well. The ophthalmoscopic examination revealed the anterior media clear, but the posterior half of the vitreous chamber filled with a white mass, with a few blood vessels coursing over the surface and many smaller ones in the substance.

After the second eye became affected, the diagnosis of bilateral glioma was made. On January 23, 1925, I enucleated the first eye to become involved. After fixation in Zenker's fluid, the globe was sent to the Army Medical Museum for pathologic study. A report of glioma was made to me, with a statement that a section had been mailed to Dr. Verhoeff of Boston for confirmation of the diagnosis.

The following report of Dr. Verhoeff's was later forwarded to me from the Army Medical Museum: "Diagnosis: Socalled glioma retinae completely filling vitreous chamber. Almost complete necrosis of tumor. Phthisis bulbi due to inflammatory reaction resulting from toxins of necrotic tumor. Optic nerve apparently not involved, but has been so greatly crushed that this can not be determined with absolute certainty."

I should like to add to this report, that the section shows considerable newly formed connective tissue adjacent to the choroid and blending with it, and a complete absence of any remains of retina. Here and there, scattered in this connective tissue are frequent masses of clumps of glioma cells. The necrotic areas are out in the vitreous, internal to this connective tissue. Only in a very few places are the cell masses

found in the choroidal layer, and at no point external to the pigment.

There are no glioma cells in the anterior chamber, and no evidence of a perforation having occurred. There is some small round cell infiltration in the external layer of the choroid.

Immediately after the diagnosis came from Washington on the eye first removed, the other eye was enucleated on February 19th, 1925. There was no atrophy of this globe.

It has been eight months since the second operation. There has been no indication whatever of a recurrence. The child is developing unusually fast and has every appearance of having perfect health.

J. Herbert Parsons[1] reported a case of glioma retinae in a shrunken globe in March, 1905, and collected from the literature fourteen other cases which had been reported previously. In his discussion of the cases he says: "In several of the cases there is no doubt that the eye became perforated before the onset of the shrinking, usually the result of the extraocular extension of the growth. In other cases it is almost impossible to decide whether perforation occurred or not." * * *

Parsons states further that the cases reported by Tyrrell, Rolland, Knapp and Grolmann are particularly interesting in that they show alternate tendencies to shrink and to grow.

Dennis F. O'Connor[2] reported a case in 1917 of atrophy of the globe with glioma of the retina, and he raises the question, "Whether the phthisis bulbi or the glioma was the primary condition." He gathered from the literature that, "the ability to produce atrophy in an eye afflicted with glioma, as deduced from the case histories and the treatment advocated by ophthalmologists of a hundred years ago, was evidently an article of faith among them," and he mentions some drugs formerly used for that purpose, among them mercury.

In reporting a case in 1920, Arnold Knapp[3] had this to say: "Occasionally the eye affected with glioma shrinks: according to Leber, the shrinkage is not due to the regression of the glioma, but is caused by an intermediary plastic uveitis, which then ends in phthisis bulbi. The cases in which this occurs and no living glioma cells remain are unusual. Berrisford found twenty cases of glioma in shrunken eyes recorded in literature, and published one case. Knieper reported on a case where shrinkage of an eye followed glioma retinae and was

under observation for a period of eleven years. * * * Reviewing twenty-two cases found in the literature, this author concluded that the period allowed for observation of the shrinkage was too short, and in these the possibility of a subsequent growth could not be excluded. Lindenfield reported the case of a child of four, with glioma in the right eye and atrophy in the left, which had first been enlarged, red and painful. This was abruptly followed by shrinking of the eye and the pain ceased, the eye remaining small for two years. The right eye enlarged, and extraorbital growth occurred; both eyes were enucleated and the shrunken eyeball showed a connective tissue mass with calcareous deposits, bone formation and no trace of living glioma cells. This case showed 'spontaneous cure' by complete necrosis of the tumor and shrinkage of the eye."

The Encyclopedia records a case of atrophy and the patient was well five years later. It was then enucleated as a precautionary measure. This was a case report by De Kleijn[4]. Quoting from the Encyclopedia: "Microscopic examination showed the characteristic features of atrophy of the bulb. The sclera contained a partly bony, partly calcareous mass in which, here and there, portions of necrotic tissue showing traces of a small celled structure occurred. The retina could not be seen, and the optic nerve was completely atrophied."

"Although microscopically it was not possible to demonstrate glioma tissue in the second eye, the author considers the diagnosis of glioma well justified and attributes the outcome of the case to some unknown factor which, in the contest between tumor and its host, turned the scale in favor of the latter."

In his review of cases reported Parsons states: "There have often been inflammatory symptoms preceding the phthisis bulbi, and it is probable that the shrinkage is due to this rather than to any inherent tendency in the growth itself (Brailey), comparable with atrophic scirrhus; such an occurrence must be so rare as to almost negative the hypothesis."

* * *

This inflammatory feature was extremely marked in my case, but with an absence of evident cause. Again the question arises as to the primary condition. Was the inflammatory condition with subsequent phthisis bulbi secondary to

tumor cell necrosis, or was the inflammatory process a coincidence and of other origin, with the phthisis bulbi and tumor cell necrosis secondary to this?

The former hypothesis would seem to be the most reasonable. Wells in his Chemical Pathology suggests: "Probably because of the great amount of necrosis that is constantly going on in all malignant growths, with subsequent digestion of the dead cells, autolytic products are present in them in very considerable amounts * * * these areas undergoing autolysis yield substances that are hemolytic and toxic." These toxins in my case from the tumor cells were probably responsible for the acute inflammation and later phthisis bulbi.

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DISCUSSION.

DR. WILLIAM C. FINNOFF, Denver: I am very much interested in the paper that has just been read by Dr. Guyton, because a few weeks before I received the second eye that he enucleated, a pair of globes had been sent to me for microscopic examination which had been enucleated from a child that presented a similar history to the case that Dr. Guyton reported.

I found in sections of the atrophic globe a few deeply stained cells in the center that were remnants of a preexisting glioma. The diagnosis accompanying the history was phthisis bulbi of the right eye following uveitis and glioma of the left.

Flexner, Wintersteiner, Collins, Parsons, Fuchs and a number of others have reported cases of atrophy of the globe in which glioma had existed. There are cases on record in which glioma had been found and, because early operation was refused, the course of the disease was observed for a varying period of time. In some of these cases, uveitis occurred and was followed by atrophy of the eyeball. In one case that presented this history, extension had occurred into the orbit, where the tumor progressed but the ocular growth became quiescent.

It is well known that bilateral glioma is of frequent occurrence. This fact should always be borne in mind, because cases are not always seen in the beginning of the disease, and it is only after the loss of vision in the supposedly good eye, that the parents are sufficiently concerned to consult an ophthalmologist. Uveitis might have occurred and been followed by atrophy of the globe, and overlooked by unobserving parents. The

former history would then be ambiguous. This occurred in the case that I have just mentioned.

The point that I wish to make is this: In cases of glioma in one eye and an atrophic globe on the other side, our suspicion should be aroused of the possibility of a preexisting gliomatous tumor in the atrophic eye with extension into the orbit. The atrophic eye should be enucleated with some of the tissue of the orbit that surround it, and sent to an eye pathologist for microscopic examination.

Sarcoma occasionally undergoes degeneration, and the toxic products that are liberated following cell necrosis excite a uveitis that may result in shrinking of the tumor and spontaneous cure. There is no reason why the same thing might not occur in glioma and produce the clinical picture that has just been described by Dr. Guyton. This probably occurred in the shrunken eye that was sent to me.

Dr. Guyton sent the eye with glioma for microscopic study. It was embedded in celloidin and sectioned. Sections were stained with hematoxylin and eosin. Microscopic examination revealed a tumor mass that occupied the greater part of the vitreous cavity. It sprang from the posterior layers of the retina in the region of the optic nerve. The remaining retina was detached and pushed both forward and laterally. The tumor covered the surface of the nerve in front of the lamina cribrosa, and extended forward into the vitreous space to within three mm. of the lens. It had not entered the choroid. There were numerous typical rosettes in the sections. The lens was in its normal position, and there was no evidence of glaucoma as the angles of the anterior chamber were normal in depth. The tumor was a typical glioma.

S. Hanford McKee, Montreal: Intraocular tumors are rare. There are two principal varieties; sarcoma of the choroid and glioma of the retina. Sarcoma occurs in adults between 40 and 60 years, is always primary, involves one eye only, and is usually pigmented.

Glioma is a disease of childhood. Of 467 cases, 314 were in the first three years, 62 in the first four years, and 29 in the first five years. No case was after the sixteenth year. In 25 per cent both eyes were involved, and it is often found in members of the same family. Lerche saw four cases among seven brothers and sisters. Wilson met with a family of eight, all of whom had neuroepitheliomata. The diagnosis is important, as early operation in these cases offers a fair chance of cure (13 per cent of recoveries). The first symptom is usually "amaurotic cat's eye". Examination shows a growth covered with a plexiform network of vessels, and it has a nodulated or knobbed surface. It must be differentiated from:

1. Simple detachment of the retina.
2. Leucosarcoma of the choroid.
3. Tubercle of choroid (history here important).
4. Chronic inflammatory process in choroid or ciliary body.
5. Acute suppurative hyalitis.
6. Cysticercus in the vitreous.
7. Congenital abnormalities.
8. Retinitis circinata.
9. Detachment of retina with dropsical degeneration of visual cells (very similar according to de Schweinitz and Shumway.)

Glioma was first recorded by Hayes in 1767. Wardrop, in 1809, gave the first precise clinical, and Robin, in 1854, the first accurate histologic description. It forms about .94 per cent of all diseases of the eye.

Microscopically it is a soft, almost fluid tumor of milky whiteness, with blood vessels and hemorrhages well marked. In stained sections, a characteristic appearance is that the stain is taken up in convoluted lines and dots, separated by masses which take up only the protoplasmic stains. The convoluted lines are seen to consist of circles and cylinders of thickly grouped cells, arranged in layers around and along axial blood vessels. Between these perivascular sheaths, are necrotic or fatty degenerated cells, which stain only diffusely or not at all with nuclear stains, especially well seen in hematoxylineosin sections. There, the blood corpuscles in the vessels stained with eosin form a center surrounded by a ring of deep blue nuclei, outside of which are necrotic areas which stain red, with or without transition zone of purple. The cause of this structure is the progress of the growth in the immediate vicinity of the newly forming blood vessels. The necrotic areas are due to insufficient nourishment of the rapidly growing cells at a short distance from the vessels, and this explains the transition zone. Wintersteiner says the tumor cells are dependent upon the vessels for nourishment: as the latter grow, they draw their cellular mantle after them, pushing the neighboring tissues apart, whereby it often happens that they come into immediate contact with necrotic areas.

In spite of previous observations, gliomas are relatively poor in blood vessels, which consist merely of endothelial tubes with a thick cloak of connective tissue, in which muscle and elastic fibers are always absent. Regressive changes are common and consist of hyalin degeneration, calcification of the vessels, thrombosis, endarteritis and periarteritis. Its origin is definitely from the granular layers of the retina. In all published cases the tumors were multiple.

Extension in the retina takes place by (1) proliferation of the primary growth, and (2) deposition of local metastasis, which are gradually overtaken and absorbed by the primary growth.

The following slides were shown:

SLIDE 1. From a child two years old. March, 1921, mother noticed three weeks previous to the consultation, that the child apparently did not see her "ball" so well. Double detachment of retina. On right side detachment complete. Double enucleation, March, 1921. Child at present well. Going to school.

SLIDE 2. Child three years, double glioma.

SLIDE 3. Child, three years, double glioma.

SLIDE 4. Sarcoma of choroid. Adult, male; note pigment.

SLIDE 5. G. F., two and a half years old. Two months previous to observation, had scarlet fever, followed by cat's eye. Vision nil. Enucleation advised. Pseudoglioma.

DR. CHARLES LUKENS, Toledo, Ohio: I reported a case of binocular glioma of the retinae at the Louisville meeting of the Academy, in 1907. This was an adult, both eyes enucleated about one year apart, the last one removed three years before the case report. Slides of each eye were shown in the meeting, and the diagnosis was unquestioned. There had been no recurrence in the three years, and there has not been in the last

eighteen years, making twenty-one years since the first operation, emphasizing Wintersteiner's contention of no recurrence if the eye be removed while the tumor is intraocular, the second eye growth being *de novo*.

Glioma of the retina is not hopeless if the treatment is radical. Such cases as these bring up the question of a double enucleation in a child. There is a terrific sentiment encountered in some places against it. The situation is often difficult for the surgeon to meet in private practice, but the professional duty is plain. With the blind schools, vocational training and the cooperation of the blind commissioners commercially with vocational products, such a mortal, living, may be an asset to his family and to the community, and not a dead weight on charity and an addition to the army of mendicants.

Dr. William L. Benedict, Rochester, Minn.: About one-fourth of the cases of glioma of the retina reveal bilateral involvement (Collins and Mayou). It has been reported as occurring in several members of the same childship, and there are three cases reported in the literature of glioma of the retina occurring in children whose parents had eyes enucleated in infancy for a similar trouble.

So far, I have no reference to the occurrence of glioma simultaneously in twins. One case reported by J. J. Pattee occurred in a twin boy, aged three years, but the other twin remained well during the nine months he was under observation.

There are ten cases of glioma retinae which have been treated with radium reported in the literature. The disease was bilateral in six, and unilateral in four of these cases. The immediate result from the use of radium was, on the whole, good. Duncan reported three cases of unilateral glioma which were treated with radium after enucleation. His second case had orbital involvement, which was not removed at the time of enucleation. X-ray failed to check the growth, but radium caused it to disappear within a month. This patient received three treatments of 50 to 110 milligrams.

Chase reported a case of bilateral glioma retinae. The left eye was enucleated and showed involvement of the nerve and paraneural sheath. The right eye showed a yellowish grey tumor, which completely covered the disc. The left orbit and right eye were each treated with eight hour applications of 30 milligrams of radium for three treatments, and four months later, there was no recurrence in the orbit and the tumor in the right eye had not increased in size. Schoenberg also reported a case of bilateral glioma retinae. The left eye was enucleated. The right eye had three treatments with radium, which caused necrosis of the glioma.

Janeway reported a case which was apparently cured by enucleation and radium treatment, and another case of bilateral glioma retinae in which the left eye was removed, and the vision of the right eye became worse in spite of heavy radium treatment.

The latest result reported was one of three years postoperation. This was Knapp's case of bilateral glioma, in which the left eye was removed with no subsequent recurrence. The right eye became involved with glioma five years later, and radium treatments seemed to improve the condition for a while, but subsequently there was a distinct failure of vision, and the eye had to be enucleated. Janeway also reported a case

of bilateral glioma, which was unsuccessfully treated with radium, the child dying from cerebral metastases about a year later.

There were two cases in which late reports were favorable. Duncan's first case of unilateral glioma with involvements of the optic nerve showed no evidence of recurrence in the orbit eighteen months after radium treatment. He used three applications of from 50 to 125 milligrams radium. Axenfeld reported a case of bilateral glioma retinae, in which the right eye was enucleated and the left eye treated with radium, with recession of the tumor and good vision after a period of three years.

I wish to report the occurrence of glioma of the retina in twin girls, at four years of age, one of whom died as a result of extension of the tumor to the brain. The other girl had glioma in each eye. The left eye was enucleated. The right was treated by radium with regression of the tumor.

Charlene and Charlotte G., twin girls, aged four years, were brought to the Clinic, November 3, 1922, referred by Doctor Lambert of Galesburg, Illinois. In March, 1922, the parents noticed a small white growth in the lower nasal quadrant, just back of the pupil of Charlotte's left eye. It gradually grew larger, and at intervals, the eye became red and the child suffered severe pain. A severe attack in July lasted eight days. The parents kept careful watch of Charlene, and late in August of the same year, a white growth appeared in the lower nasal quadrant of her left eye, and steadily increased in size. She occasionally complained of sharp pain of short duration.

On examination of Charlotte G., a mild photophobia was found. The ocular conjunctiva of the left eye was injected, the cornea was clear, and the anterior chamber was very shallow. The pupil was so widely dilated, that only a rim of the iris could be seen. As the lens was opaque and the eye transilluminated very poorly, no details of the fundus could be seen. No lesion of the fundus of the right eye was detected. When the child was seen the next day, photophobia was marked, the left eye was painful, and there was a yellowish reflex from the dilated pupil.

Charlene G. had no subjective symptoms. Externally, the eyes were negative except that a grey-white mass, 7 mm. in diameter, projected from behind the iris in the lower nasal quadrant of the left eye. The ophthalmoscopic changes were conspicuous. There were many floating opacities, both large and small, and the lower nasal quadrant showed a grey-white elevation, irregular in outline, over which the retinal vessel arborized. From this mass a few grey-white strands extended medially toward the disc. There were marked choroidal and retinal disturbances around the mass. Transillumination of this area was very poor.

The media of the right eye were clear. The nervehead was round and of good color. In the lower temporal and inferior quadrant, there was marked retinal detachment and an elevation of five diopters, gradually increasing toward the periphery. There were many small, grey-white round areas with pigment deposits around them. In the central portion of the detachment, running obliquely about axis 30 degrees, was a lighter red area, irregular in shape. The retina was detached in the lower half of the fundus, and varied in height, width, and amount of retinal change.

A clinical diagnosis of glioma of the retina of the left eye was made for both children and immediate enucleation advised. It was impossible

to determine whether the detachment in Charlene's right eye was due to the same cause, but it was at least quite probable.

Both left eyes were enucleated December 7, 1922. Pathologic examination of Charlotte's left eye was reported as follows: Anterio-posterior diameter, 26 mm.; equatorial diameter, 25 mm. The cornea is round, smooth, transparent, 11 mm. in diameter. The pupil is dilated ad maximum. The contents of the anterior chamber have a yellowish color. The optic nerve is enlarged, 7 mm. in diameter, soft and fleshy in appearance, and where sectioned, the contents bulge over the edge of the optic sheath. There is some fatty tissue adherent around the optic nerve entrance.

The globe was sectioned in the horizontal plane (without freezing) through the optic nerve. A milky fluid escaped, in which were small white flakes. The anterior chamber contained a small amount of the fluid. The iris is contracted to a narrow band, the lens is in place, but the nucleus is white and opaque. The optic nerve and sheath and the posterior one-third of the vitreous chamber are filled with a firm white tissue, which is discolored brown in places. The rest of the vitreous chamber is filled with a soft fungating mass, creamy white in color and opaque. It seems to arise immediately over the optic disc. One-half of the eye was mounted in gelatin for a museum specimen. The other half was dehydrated in alcohol, embedded in celloidin, cleared in cedar oil, cut dry, and stained in hematoxylin and eosin for cytologic examination. The pathologic diagnosis of the section of the globe was "cellular glioma" of the optic nerve, "degenerating nerve (no glioma)". After an uneventful convalescence, she was dismissed to return to her home.

On February 8, 1923, she returned with no evidence of recurrence, and was given an artificial eye. March 19, 1923, she again returned, because for three weeks she had complained of pain over the left eye and had vomited several times daily. She was greatly emaciated. The right eye was normal, and there was only slight fullness of the left orbit, no definite mass. On March 30, 1923, under general anesthetic, the orbit was explored, and a piece of tissue removed for examination. The pathologic report of this tissue removed was "cellular glioma." She was then given 2038 milligram hours of radium at one sitting, over the left orbit.

She was again brought to the Clinic on May 4th. Following the radium treatment, she gained in weight and seemed much better until the last few days, when she began to vomit and to have a great deal of pain in the head. The tumor within the orbit had increased in size until it protruded between the lids. The child had mild Jacksonian attacks in the right side and persistently refused food. She grew more stuporous, and died of respiratory failure on May 25th. At autopsy, recurrence of the tumor was found in the orbit, with extension into the cranial cavity, hemorrhagic softening of the frontal lobe, internal hydrocephalus and terminal hemorrhagic bronchial pneumonia.

Pathologic examination of Charlene's left eye was as follows: The globe is about normal in size and shape. The anterio-posterior diameter is 22 mm.; equatorial diameter, horizontal, 23 mm.; equatorial diameter, vertical, 22.5 mm. The cornea is clear; the anterior chamber regular, equal depth, but somewhat shallow in the lower temporal quadrant. The iris is grey; pupil regular, slightly oval; lens translucent. The sclera is blue except for an area about 7 mm. wide between the insertions of the

rectus internus and inferioris, where it is white and more resistant to pressure. The optic nerve was sectioned in the horizontal plane without freezing. The anterior chamber is partially filled with a very soft white mass, which fairly well fills the nasal one-half and extends forward to the posterior surface of the lens. It also lines the posterior portion with a thick layer, leaving only the temporal side of the retina visible in front of the equator. Otherwise, the chamber is filled with cloudy white fluid.

The globe was sectioned through the cornea and optic nerve, and one-half of the specimen preserved in gelatin. The pathologic diagnosis was "glioma retinae".

Ophthalmoscopic examination of the right eye revealed, in the lower temporal and inferior quadrant, a subretinal mass with elevation of 5 diopters, over which the retina was detached. There were many small, greyish-white, round areas surrounding the mass, with some pigment changes. In the central portion of the mass, running obliquely about axis 60, the retina was lighter red in color and irregular in shape. The detachment extends from about 5 to 9, and varies in size and height and character of change. The central portion was more pale. Knowing that the mass in the left eye was glioma, it was felt that there was also a glioma in the right eye, and I decided to use radium in an attempt to reduce the mass. From December 12, 1922, to October 19, 1924, a total of 40,000 milligram hours of radium was applied with suitable screening to the right eye. There was a gradual diminution in size of the mass, with gradual destruction of the area involved. On October 16, 1924, the ophthalmoscopic examination showed the nasal portion of the retina to be of normal appearance. In the lower temporal quadrant, there was a large pigmented area over the site of the previous detachment. The retina had now become reattached except in the lower quadrant, where there was a small domelike detachment, greyish-white in color, with numerous small hemorrhages. With correction, sphere plus 2 combined with a cyl. .50 axis 90, vision equaled 6/20.

Dr. George Slocum, Ann Arbor, Michigan: We have now under treatment and observation in the clinic of ophthalmic surgery, in the University of Michigan, a case which was first seen a little over a year ago. The diagnosis was not difficult. There was a typical socalled glioma in one eye. Ophthalmoscopic examination of the other eye revealed a mass in the inferior mesial region, two or three times the size of the disc; and in the superior mesial region, a second mass about twice the size of the other. The child was about two and a half or three years old. Because of the good vision present in the eye which was the least involved, only one eye was removed. The other eye has since been treated with deep X-ray therapy, and there has been no change in the condition since the treatment was instituted. I saw the case the last time about a month ago.

Dr. George F. Keiper, Lafayette, Indiana: These tumors of the eye arise in the neural epiblast (teleplasms) and are either pigmented or non-pigmented. To this latter class belong the tumors under discussion. These tumors have been unfortunately named, and we therefore should abandon the name "glioma" of the retina, that we may not be confused with glioma of the brain, which is an entirely different proposition. They should be called neuroepitheliomata. The tumor may arise either from the optic

nerve, the ciliary processes, or the nerve cells of the retina itself. I exhibit a specimen showing all these origins. The tumor may grow outward from the retina, and is called glioma exophytum. It may grow inward, and is called glioma endophytum. This specimen also shows these characteristics It may extend backward through the optic nerve to the brain. Microscopically, there is nothing like it in the arangement of the cells into rosettes.

Dr. McKee has touched upon the question of age. I have never seen a case later than eleven years of age. The two eyes may be affected, and

Glioma of the Retina.

an interval of time usually elapses between the two—as much as three years, according to a case of Mr. Treacher Collins. The two eyes are thus affected in one-third of the cases. The two sexes are equally affected.

This disease may be mistaken for buphthalmos, with yielding of the ocular tunics on account of increased tension. But let us give the disease its right name—neuroepithelioma and not glioma.

Dr. James M. Patton, Omaha, Neb.: I wish to mention a case which is in contrast to the one presented by Dr. Benedict, and incidentally to urge the advisability of instructing our colleagues in the general profession, and our students, in the importance of making an early diagnosis of this condition.

This case was a child, 18 months old, who was brought to us within the last two or three weeks with both globes three-quarters full of what was undoubtedly glioma tissue. The child had been suffering for six months, with a diagnosis of congenital cataract, and the parents had been told there was no hurry about an operation—to wait—that the cataracts could be removed at any time. We have no way of knowing what the

condition of these tumors was six months ago, when first discovered, but when we see the result which Dr. Benedict has been able to secure from the use of radium, it is entirely possible that vision might have been saved if the child had been given prompt attention. I think, as ophthalmologists, we should take every opportunity to impress upon groups of general physicians the necessity for prompt action in all cases that might in any way be diagnosed as glioma.

Dr. E. C. Ellett, Memphis, Tenn.: Dr. Guyton's patient was shown before the Memphis Eye and Ear Society, when I had the opportunity to examine the eyes. I had not seen a case before in which the eye and tumor had shrunk from the beginning, but I would like to mention two cases of bilateral retinal glioma which I have had under observation, and which presented some interesting features.

Case 1. B. J., aged one year, was seen in April, 1921, with bilateral retinal glioma. Radium was given a thorough trial, but the right eye, growing apparently worse, was removed in September, 1921. The left eye was subjected to more radium, without effect, and it was removed in July 1922.

Case 2. M. M., aged six months, was seen in 1922 with retinal glioma, O. D., and the eye was removed. Between the examination and the day of operation—three days—the aqueous humor became so clouded that the pupil could not be seen. This was found to be due to the presence of tumor cells in the anterior chamber (Pseudohypopyon). Under the anesthetic, the left eye was carefully examined with the pupil dilated, and a round white mass was seen 3 d. d. wide, elevated about 15 D. from the level of the retina, 4 d. d. below the disc and a little to the temporal side. No vessels were seen on this tumor, which looked like a big snowball lying on the retina. This eye was treated by radium, 1000 millicuries being given, without effect. The tumor continued to grow and the eye was removed in June, 1925. The gross appearance was that of glioma, but the histologic study has not been completed. (Slides from the right eye of the last patient and of both eyes of the first one were shown.)

Dr. B. S. Guyton, Oxford, Miss., (closing): In presenting this case, I meant to emphasize the fact that there was atrophy of the globe in one eye. The literature states that we may have bilateral glioma in 25 per cent. of all cases, but so far only about 25 cases are on record in which there has been atrophy of the globe in glioma.

PROBLEMS CONNECTED WITH THE SURGICAL TREATMENT OF OCULAR PALSIES.

JOHN MONRO BANISTER, A. B., M. D., F. A. C. S.

OMAHA, NEB.

I. GENERAL CONSIDERATIONS.

This paper, as its title indicates, is not presented as an academic discussion of paralyses of the ocular musculature, but simply as a practical surgical contribution, embracing in its limited scope certain of the problems which the ophthalmic surgeon must face when cases of paralysis of this type are brought to him for relief. Consequently, the enumeration of the many causes which may be responsible for palsies of the ocular muscles, with a review of ophthalmologic literature bearing upon such causes and their diagnosis, would be out of place in this essay.

These conditions can be found thoroughly discussed in the text-books. There is one set of causative conditions, however, to which reference should be made, and which has received little attention from ophthalmologic writers, as a rule, and that is the group of focal infections, which may have an important influence in inducing paretic states of the ocular muscles, generally of a temporary character. The fact should also be mentioned, that syphilis is responsible for about fifty per cent of the cases—a valuable diagnostic and therapeutic hint.

In advance of operation, the practical surgeon must eliminate from the problem the conjugate palsies, indirect paralyses, and the presence of progressive intracranial disease of whatever type, as such states mark any case as inoperable on account of the underlying pathology.

The cases offering prospects of success following operation must be limited to direct paralyses of individual muscles resulting from lesions affecting especially the nerve trunks, or the individual branches to special muscles. A lesion of an oculomotor nucleus is likely to affect both eyes, since each

nucleus sends fibers to both sides[1]. Nuclear palsies, therefore, introduce an element of doubt.

Paralyses of individual muscles will never result from "lesions of the centers of higher rank situated above the nerve nuclei (that is the cerebral cortex, the association centers, and the fibers connecting these parts with one another and with the nuclei)[2]" which will invariably cause conjugate paralyses where the "eyes are unable to turn in some special direction, or they may be unable to converge or diverge" (Duane[3]), which states are inoperable from the nature of the pathology involved.

Furthermore, the cases of direct paralyses of individual muscles, constituting the operable class, must have been proved to be permanent by the unsuccessful application of efficient means of treatment, addressed to the pathologic states responsible, for at least six months, preferably longer, before the advisability of operation can be decided upon. In considering these cases, and in devising the most appropriate surgical treatment therefor, an important physiologic law, applicable to all of the muscles of the body, must be given its full weight, namely the law of "Adaptive muscular shortening."

"The essential property of muscular tissue is contractibility, shown by an ever present tendency to shorten. Every muscle in the body is in a condition of slightly continued contraction, which keeps it tense. * * *

If a muscle is kept any length of time in such a position that its ends are brought nearer each other, it still remains in tone by shortening." If this condition should continue for a longer period, a real shortening will occur, and "the longer the necessary fixation, the greater the adaptive shortening."

These statements are quoted from Orthopedic Surgery, Jones and Lovett, a standard work in the special field of orthopedics, and apply to the ocular muscles, as well as to those in other parts of the body.[4]

The orthopedic surgeon makes use of tenotomy in relieving the results of such contraction. Why should not the oculist do the same in cases of paralytic strabismus, where the unopposed antagonist may be considered to be always in a state of greater or lesser contraction in accordance with the general physiologic principles just stated?

The writer is of the opinion that tenotomy of the antagonist in cases of paralytic ocular deviations, in addition to

the required attention to the paralyzed muscle in the way of advancement or operative shortening, is essential to success in the great majority of cases. Indeed, the elimination of the unknown quantity from the operative equation, in this case the degree of acquired shortening, or secondary contraction, of the antagonist, is necessary for the solution of the surgical problem, as both mathematics and physiology teach us.

A very practical advantage resulting from the tenotomy of the antagonist is this, that after its performance, a seemingly completely paralyzed muscle will frequently be found to have quite an appreciable degree of contractility, previously neutralized by the contraction of the antagonist, the releasing of which remnant of power will be of great value in securing the best result from operation.

In fine, after considering these general phases of the problem, it may be said in a condensed way, that the cases with which the ophthalmic surgeon will be called upon to deal, are the direct palsies of individual muscles where there are no conditions dangerous to life, where the causative lesion is not a progressive one, where the paralysis is permanent and fixed, and where the patient is not disqualified for the performance of his duties except by reason of the distressing diplopia and deformity existing.

Under such conditions there is no place for professional agnosticism or surgical hesitation.

II. Special Operative Problems.

1. Paralysis of the internal rectus muscle with outward deviation of the eye and heteronymous diplopia: In this condition the following solution of the operative problem is advised: First perform a complete tenotomy of the externus (antagonist) and, preferably at the same time, follow this step by an advancement, or shortening, of the paralyzed muscle to such a degree, that the visual lines of the two eyes will be parallel when the sound eye fixes a test object at twenty feet.

Even in cases of complete paralysis of the internus, this operative treatment may be expected to secure single vision for points directly to the front and throughout the sphere of action of the externus, which latter muscle will still be able to turn the eye well to the outer canthus.

In a surprising number of instances, however, it will be

found that there still remains in the internus a degree of contractility which can be effectively utilized after the preponderating power of the externus, due in part to its "adaptive shortening," has been curtailed by the tenotomy. This possibility has already been referred to. The writer looks upon the tenotomy of the antagonist in such cases as the key of successful treatment.

Figure 1 shows the cosmetic result obtained by the writer in a recent case of permanent paralysis of the left internus of luetic

Fig. 1.

origin, manifesting a condition of extreme deformity with distressing diplopia. Single vision was obtained for points straight ahead.

The affected eye (left) had been previously operated upon by another surgeon without tenotomy of the antagonist, and hence without effect, the outward deviation still being of marked degree and the diplopia disqualifying. It should be noted, that in certain of these cases, where the internus is not completely paralyzed, and where its remnant of power has been permitted to manifest itself in consequence of the tenotomy of the antagonist, the sound eye will strongly devi-

ate outwards, under the overstimulation of the associated externus of that eye, when the palsied eye attempts to turn toward the nasal side. This is in accordance with a well known physiologic law.

Under such conditions, an extreme degree of heteronymous diplopia is manifested, the false image belonging to the secondarily deviated sound eye.

Figure 2 furnishes a photographic illustration of such secondary deviation, in the case of the patient depicted in Figure 1,

Fig. 2.

following operation, as advised, on the left eye with paretic internus.

When this occurs a new problem arises, the solution of which from the writer's point of view should require a tenotomy of the overacting externus of the secondarily deviated sound eye, or an operation to advance, or shorten, the internus of the same eye, or both combined where necessary. A similar problem may occur in connection with paralyses of other ocular muscles.

As the result of this operative treatment, the field of single

vision may possibly be increased to an unexpected degree toward the side of the sound eye.

2. Paralysis of the external rectus muscle with inward deviation of the eye and homonymous diplopia: The problem presented in such a case, which demands the relief of the deformity and the securing of single vision for points straight ahead, may best be solved by the performance of a tenotomy of the internus (antagonist), for reasons previously stated, followed by an advancement or shortening of the paretic externus to the extent of procuring parallelism of the visual lines for twenty feet.

Advancement of the paralyzed externus without previous tenotomy of the antagonistic internus will, in all likelihood, fail to secure the desired result, owing to the "adaptive shortening," or secondary contraction, of the latter muscle.

Then, as before stated, tenotomy of the antagonist may serve to release some remaining, though masked, power residing in the externus in cases where the paralysis is not complete—a fact of decided advantage.

3. Paralysis of the superior rectus muscle with downward deviation of the eye, and with vertical and heteronymous diplopia. The images are crossed, with the false image in the upper visual field: The surgical solution of the problem offered by this condition has given rise to quite an amount of controversy.

Some ophthalmologists have advised a technical advancement of the paralyzed muscle, to bring the cornea upon a horizontal level with that of the sound eye when the eyes are directed straight forwards.

The objection to this method is, that after its performance, there is a likelihood of the occurrence of diplopia in the lower visual field, as Dr. Duane has so pertinently insisted upon. The reason of this is that in the technical advancement, the divided tendon is reattached to the globe nearer to the cornea, which gives an exaggerated mechanical pull upward; for, the nearer to the cornea the attachment, the greater the pull. This upward pull is likely to overmaster the inferior rectus and curtail its proper function to a degree sufficient to cause diplopia in the lower field.

If, on the other hand, a tuck shortening is applied to the superior rectus, the portion of the belly of the muscle em-

braced in the tuck is brought up to the normal insertion and there fixed.

This method of shortening has never in the writer's experience been sufficient to produce the overeffect just referred to, with diplopia in the lower field. Indeed, after its performance, in order to secure the proper leveling of the two eyes, the inferior rectus must be set back to a sufficient degree and there fixed. The writer secures this effect through what may be called a controlled tenotomy, after the following method.

The tuck is first taken in the paralyzed superior rectus, and even with the full effect of such performance, it will be found that the cornea is still directed downwards to some degree, and

Figure 3. Figure 4.

Figs. 3 and 4.

that there is still diplopia in the upper field. Then the tendon of the inferior rectus is exposed, its attachments to Tenon's capsule being interfered with as little as possible, and a loop of 10 days, chromicized, "00" catgut is passed through the belly of the muscle about 4 millimeters from the insertion. A complete tenotomy is then performed as usual. The tendon will slip back, and it will be found that there has been secured an overeffect as regards the upward turning of the cornea, owing to the mechanical effect of the tuck, and possibly in some degree, to the latent power in the superior rectus in cases of incomplete paralysis.

Then the two ends of the suture, which has looped the belly of the inferior rectus, are passed through the remnant of the insertion of the tendon, embracing in so doing the episcleral tissue. The first turn of a surgeon's knot is then taken, and gradually tightened, drawing towards the former insertion the severed end of the muscle until the proper level-

ing result has been obtained. The knot is then completed and the divided end of the inferior rectus muscle is thus fixed at exactly the right distance from its former insertion to secure the result desired. By this method, brought forward by the writer some years ago, excellent results may be obtained and the danger of diplopia in the lower field reduced to an improbable minimum. Figures 3 and 4 will illustrate the method described.

The imminent danger of diplopia in the lower field after the technical advancement of the paralyzed superior rectus has caused so eminent an authority as Dr. Duane of New York to advocate and practice the performance of a tenotomy of the contralateral inferior oblique muscle of the sound eye.[5]

Fig. 5. Paralysis of left superior rectus.

Fig. 6. Paralysis of right inferior oblique.

The superior rectus of one eye and its contralateral muscle, the inferior oblique of the other, are closely associated throughout the entire range of motion of the two eyes. If the *left* superior rectus, for instance, is paralyzed, a heteronymous and vertical diplopia will result, the false image being to the right of the true image and displaced upwards with its upper extremity rotated to the right. In paralysis of the contralateral muscle, the inferior oblique of the *right* eye, the diplopia is homonymous and vertical, the false image being to the right and displaced upwards with its upper extremity rotated to the right. In simultaneous paralysis of the superior rectus of one eye and the contralateral inferior oblique of the other there should (theoretically) be no diplopia, since the images of the two eyes will then occupy similar positions in space, manifesting a parallel obliquity, and will be able to be fused.

This is the result that Dr. Duane has aimed to secure by tenotomizing the contralateral inferior oblique of the sound eye.

In considering this method the following points may be noted: (1) A sound muscle pertaining to a sound eye is sacrificed to compensate for the deficiency in the paralyzed muscle of the affected eye. (2) The inferior oblique is divided at its origin, and is definitely paralyzed so that the effect of its section cannot be regulated. (3) It would seem better, therefore, to secure the desired result by devoting attention to the offending eye if it be possible to relieve the diplopia in this way.

Figures 5 and 6 are explanatory, and show the relative positions of the images of the two eyes, when, in paralysis of the superior rectus of the left eye, the contralateral inferior oblique of the sound eye is tenotomized in accordance with the method of Dr. Duane.[6]

Another problem connected with paralysis of the superior rectus may possibly at times obtrude itself upon the attention of the oculist, namely, what should be done to correct the upshoot of the sound eye to the nasal side when the paralyzed eye attempts to fix and look outwards, as may sometimes happen?

This upshoot is evidently due, in accordance with the physiologic rule, to an overaction of the elevators of the sound eye in consequence of the overstimulation of these muscles, the superior rectus and inferior oblique, resulting from the extreme effort to elevate the paretic eye in the act of attempted fixation. This upshoot has been credited chiefly to a spasmodic contraction of the inferior oblique, and a tenotomy of this muscle proposed as a corrective.[7] The superior rectus, however, has a part to play, also, in this upshoot, and a division of the tendon of its fellow elevator, the inferior oblique, cannot alone be expected to bring the two eyes on a horizontal level and relieve the diplopia due to the downward inclination of the paretic eye when the sound eye fixes.

Would it not be better to devote attention to the palsied side after the method advised by the writer in the preceding paragraphs?

By this method, which is eminently practicable, the upshoot of the sound eye would be relieved for physiologic reasons, resulting from the surgical elevation of the palsied organ, and this elevation would obviate the vertical diplopia with the false image in the upper field.

4. Paralysis of the inferior rectus muscle with upward

deviation of the cornea and with vertical and heteronymous diplopia. The images are crossed, with the false image in the lower visual field: The problem, here, is reversed, and the writer advises its solution by a method similar to that advocated in the case of paralysis of superior rectus, namely a shortening of the inferior rectus and a controlled tenotomy of the superior of the same eye.

5. Paralysis of the superior oblique muscle with vertical and homonymous diplopia, the false image being directed downwards and outwards, and its upper extremity rotated inwards: In considering the solution of this problem, it is evident that, for anatomic reasons, any operative measure of the nature of a shortening or advancement, applied directly to the paralyzed muscle, would be out of the question. Therefore

Fig. 7. Paralysis of left inferior rectus.

Fig. 8. Paralysis of right superior oblique.

the relief of the diplopia must be sought in a setting back of the contralateral inferior rectus muscle of the sound eye, which operation offers the best chance for reasons fully discussed. Figures 7 and 8 will serve to illustrate.

6. Paralysis of the inferior oblique muscle with homonymous diplopia, the false image being directed upwards and outwards, and its upper extremity rotated outwards: As in the case of paralysis of the superior oblique, operation upon the inferior oblique itself, as a corrective measure, is impracticable. The operative problem can best be solved in such case by setting back through a complete tenotomy, the contralateral superior rectus muscle of the sound eye, which surgical measure will cause the images of the two eyes to occupy similar positions in space and to assume a corresponding obliquity permitting of fusion. (See figures 5 and 6).

7. Combined paralyses of vertical and lateral recti muscles of the same eye. Such paralyses present at times

problems of quite a degree of complexity. These problems may be solved satisfactorily, in the great majority of instances, by treating the vertical and lateral deviations separately as distinct conditions, in accordance with the methods for each already described, and limiting the treatment to the affected eye, if possible.

The writer advocates the correction of the vertical deviation first, and then, in about a week or ten days, the relief of the lateral anomaly by the appropriate operation on the externus or internus, or both, of the palsied eye, as the case may require.

Figures 9 and 10 present preoperative and postoperative photographs in a case of traumatic paralysis of the right inferior rectus and a partial paresis of the right internal rectus muscle

Fig. 9.

of traumatic origin occurring nine years ago. The affected eye was operated upon in 1923.

First the inferior rectus was shortened by a tuck followed by a controlled tenotomy of the superior rectus of the same eye. These procedures leveled the eyes in the vertical sense, leaving an outward deviation. A week later a controlled tenotomy of the externus of the affected eye was performed relieving the external deviation.

The final result is shown in Figure 10, a recently taken photograph.

The result has been perfect. The eyes now move in harmony, and the patient is doing satisfactory work at college, as she informs me in a letter just received.

Vision in the right eye has been only 20/200 owing to the

Fig. 10

habitual suppression of the image for over seven years. The patient has no diplopia.

Summary of Surgical Measures Advised.

1. Where paralyses of recti muscle occur, limit surgical attention to the straight muscles of the affected eye, if possible. If a greater effect is still required, secure such through supplemental attention to the appropriate rectus, or recti, of the sound eye.

2. In the case of lateral palsies, tenotomize the antagonist of the affected eye and then advance, or shorten, the paralyzed muscle of the same eye.

3. In the case of vertical palsies, shorten the paralyzed muscle, and then lessen the action of the antagonist of the same eye to the requisite degree by a controlled tenotomy.

4. In paralysis of an oblique muscle, tenotomize the appropriate contralateral rectus muscle of the sound eye in each instance.

5. In combined paralyses of recti muscles of one eye, treat the vertical and lateral deviations separately as distinct conditions in accordance with the methods advised.

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DISCUSSION.

DR. H. W. WOODRUFF, Joliet, Illinois: I was not aware that there was any controversy about the need of tenotomizing the antagonist in cases of paralysis, whether partial or complete, so I was much surprised to hear Dr. Banister emphasizing that point, because the need of it is apparent, not only in cases of paralysis, but often in cases of ordinary concomitant squint.

I agree with the Doctor as to his method of dealing with cases of partial paralysis. He has, however, not mentioned the cases of complete paralysis. I think these are the greatest bugbear of ophthalmic surgery. Up to a few years ago, surgeons were so intent on securing a result, that they not only advanced the paralyzed muscle to the extreme, but also resected the opponent, perhaps removing all of the tendon, so the muscle could not reattach itself. I have been through that experience and failed to get results in cases of complete paralysis. When the paralyzed muscle is called into action, for instance the external rectus, and the patient is unable to bring his eye beyond the median line, that I think is pretty good evidence that he has complete paralysis of the external rectus muscle, and if such is the case and it has existed over a period of years, it is no longer a muscle in the sense of function—it is simply a band of fibrous

Case 1. Paralysis of both external recti

Case 2. Paralysis of the left external rectus; fourteen years' duration.

Case 2. Result after partial tendon transplantation of the superior and inferior recti muscles to the external rectus.

Case 3. Paralysis of the left external rectus, six and one-half years' duration.

Case 3. Result after partial tendon transplantation of the superior and inferior recti muscles to the external rectus.

tissue and has no power of contraction at all. You might as well let it alone so far as getting any result by shortening or advancing is concerned. It is principally of this class of cases that I shall speak.

I have a few pictures of cases. The first one is shown as a curiosity. It is one of those cases of extreme convergence due to paralysis of both external recti, with contraction of the internal recti. This patient 25 or 30 years ago visited various clinics in this city and was operated upon by several men. Dr. Fisher and Dr. Wilder have both seen this patient, and Dr. Wilder thinks he has operated upon this woman. What the final result was in that case I am unable to state. The cornea in each eye was almost entirely concealed by the caruncle, so extreme was the amount of convergence. It is obvious that no method of tucking with tenotomy would permanently correct this case. I believe, however, that the method of tendon transplantation would have offered something in this case.

In 1917, I presented a paper to the Section on Ophthalmology of the American Medical Association, on the subject of tendon transplantation for paralysis. This picture, case 2, is the first patient upon whom I had attempted this operation. She had had previous operations—tenotomies and advancements—without result, and I was persuaded to attempt the operation of tendon transplantation, using the outer halves of the superior and inferior recti muscles. Partial correction was obtained.

The next picture, case 3, is a boy, seven years old, who had congenital paralysis of the external rectus. I secured perfect cosmetic result

Case 4. Paralysis of both external recti.

Case 4. Result after tendon transplantation

by that operation. Of course that is about all we can expect in these cases of complete paralysis.

This man, case 4, 60 years of age, had suffered injury by falling from a street car seven years before, and in addition to that he had a positive Wassermann, and the neurologist also diagnosed tabes. He presented most extreme convergence in both eyes, due to the paralysis of both external recti and contraction of the interni. On account of my previous experience, I was induced to attempt this operation of tendon transplantation on this patient in two operations. On the first eye, I was so intent on getting a result, that I not only transplanted the outer halves of the inferior and superior recti, but also resected the tendon of the opposing muscle, so that I secured a somewhat overeffect. So when I operated on the next eye, I confined my operative procedure to tendon transplantation and simple tenotomy of the internal rectus, with very satisfactory results in both eyes.

I have therefore come to the conclusion that even the worst cases of paralysis of the muscles of the eye can be cosmetically cured by this procedure.

Dr. Alexander Duane, New York City: Dr. Banister, in opening his paper, pointed out the importance, from an operative viewpoint, of the well known fact, that paralysis of one muscle is regularly associated with overaction of its opponent. When this overaction is marked, it affords certainly an indication for tenotomy of the antagonist to the paralyzed muscle. Generally, by a careful analysis of the way in which the deviation and diplopia alter, and the way in which each eye moves as the gaze is shifted, it is possible to determine how great the overaction is in a given case, and thereby decide if and how much we shall gain by tenotomy. This is important, for it is quite certain that in many cases tenotomy of the antagonist is not only not helpful but is disastrous. Whether to tenotomize or not should in general depend on the extent to which the diplopia and deviation due to the paralysis extend into the field of action of the opponent, and also whether and how much the range of excursion of the latter is increased. A case in which the opponent functions beyond its usual range, and in which the deviation remains considerable even after the opponent has carried the eye beyond the midline, would call for a tenotomy. On the other hand, if we do tenotomy in a case in which the deviation is practically confined to the field of action of the paralyzed muscle, and in which the excursion of the opponent is no more than normal or not in excess of that of its associate in the other eye, we shall in many cases find ourselves in the unpleasant position of having aggravated the patient's disabilities by giving him two paralyses instead of one.

There is one other point to which Dr. Banister does not advert, but which seems to me most important in explaining the effect of an operation. In paralysis, the thing that causes trouble is not the restriction of movement per se. In ordinary looking, we rarely move our eyes more than 10° or at most 15° from the midline, so that even a well marked restriction causes little inconvenience. Nor is the main trouble ascribable to the deviation and diplopia in the primary position. Rather is it caused by the fact that both deviation and diplopia keep changing as the eyes are moved. The patient soon learns to make allowance for a diplopia

which changes but little as the eyes are turned this way and that; he cannot do this for a diplopia which changes notably with every shift of the gaze, and hence is kept in a state of uncertainty and confusion. Accordingly, as my experience has repeatedly demonstrated, an operation which secures even an approximate correction produces great relief, provided it transforms the diplopia from a variable to a more or less constant quantity.

Turning now to the individual conditions described by Dr. Banister, I should like to outline the procedure which I have found useful, and which in several respects differ from those which he has given. In speaking of the operations, I should say that I employ the generic term advancement to include any operation—advancement proper, resection, tendon tucking—calculated to give the muscle better leverage; and that I use tenotomy to denote both the ordinary operation (with or without restraining suture) and Jameson's operation of recession. Of the numerous advancement operations, I have seen good results from the Worth operation, Reese's resection, the single stitch operation, and the Bruns-Clark tucking-advancement—each having its indication according to the condition to be dealt with. Thus the single stitch operation seems to me specially advantageous in case of operation on a muscle that has already been tenotomized or advanced; the Bruns-Clark operation is good if we are dealing with a fragile tendon, etc. There are many other good operations which I have not tried. Of the procedure for setting the tendon back, recession seems to me the operation par excellence for the straight muscles, particularly when a considerable effect is to be produced. I regard it as the most important advance that has been made in recent years in the operative correction of motor anomalies.

The particular conditions that Dr. Banister considers are:

1. Paralysis of the internal rectus. Here I think it is generally necessary, as Dr. Banister says, to combine advancement and tenotomy. If the evidences of overaction of the external rectus are considerable, I should try to get more and more of the desired effect by the tenotomy (recession), and in the contrary event depend more and more on the advancement.

2. Paralysis of the external rectus. Here much the same principle applies, with the difference that we must be particularly cautious in seeking to get the effect by means of a tenotomy; for if we are careless in this regard, we may cripple the internal rectus in its important function, that of convergence. A tenotomy of the external rectus, which restricts somewhat the movement of the eye outward, will cause little inconvenience, because we rarely need to move the eye over 10° or 15° at most out beyond the midline; but a tenotomy of the internal rectus which restricts the movement of the eye inwards to any great degree will interfere with convergence and may cause troublesome asthenopia in reading. If, then, we do an ordinary tenotomy of the internal rectus, I prefer to apply a temporary limiting suture. Still better is recession, as experience shows that this operation even carried to a considerable extent, rarely causes any notable impairment of convergence.

3. Paralysis of the superior rectus. Here my experience differs widely from that of Dr. Banister. Advancement of the paretic muscle, even when done by a competent operator, has produced restriction of action of the

opponent, and hence a diplopia in the lower field, where of all places diplopia is most troublesome. Moreover, the permanent effect in many cases leaves a good deal to be desired. On the contrary, I have derived great satisfaction in this condition from a tenotomy of the contralateral inferior oblique. This operation is the more indicated, the more the diplopia and deviation are confined to the upper field, and the more pronounced the upshoot of the fellow eye when its gaze is directed inward and upward.

Dr. Banister urges various objections against the operation which, I am sure, are theoretic only, for they are not borne out by the experience of myself and of my associate, Dr. White, who between us have been concerned with nearly one hundred and fifty of these operations. The sort of result that is secured is indicated in the accompanying diagram, showing the diplopia before and after operation. The patient whose case is depicted experienced very considerable and immediate relief—evidently not so much because the deviation was very greatly reduced, as because both deviation and diplopia (as the cards show) were made much more uniform through the field of fixation.

In cases of this sort in which the diplopia is carried well down into the lower field, the operation may need to be supplemented by a very guarded recession of the inferior rectus.

4. Paralysis of inferior rectus. Here advancement of the affected muscle is the best operation. In no case would I do tenotomy of the superior rectus, unless the residual deviation was considerable and also was carried well up into the upper field. Even then I should be rather reluctant to do the operation, for a number of times unpleasant results (evidences of double paralysis, i. e. of diplopia increasing both up and down) have developed in consequence.

5. Paralysis of the inferior oblique would call for tenotomy of the contralateral superior rectus. I have never seen a case of the sort that required operation.

6. Paralysis of the superior oblique may be met by recession of the contralateral inferior rectus. In not a few cases, however, there is a considerable deviation in the upper field produced by spasmodic action of the inferior oblique in the same eye; and these cases I find are satisfactorily dealt with by a tenotomy of this inferior oblique.

These are general rules, but each case offers its own problems, and its solution requires a careful analysis of all the conditions present.

Dr. James M. Patton, Omaha, Nebraska: Those of us who have been so fortunate as to be intimately acquainted with Dr. Banister, have thoroughly appreciated the many practical suggestions he has so generously given with relation to the management of ocular muscle deformities; and in the paper which we have just heard, he has emphasized a number of points which we can do well to profit by. Too often, I am afraid, we approach these cases with the idea of the deformity so firmly fixed in our minds, that we forget or neglect to consider the mechanical question involved. The theory of adaptive muscle shortening is well known, and the presence of latent power in an apparently paralyzed muscle is entirely logical. We have been agreeably surprised, at least I know I have, at the restoration of function secured by the simple tenotomy of an antagonist. In some instances, the tenotomy was preparatory to a

further shortening operation which was never necessary. I think our natural hesitancy in doing a tenotomy, due to the fear of overcorrection, can be entirely disregarded if we employ the controlled method as suggested by Dr. Banister. The technic he suggests is very simple and effective, and if properly carried out, should preclude the possibility of overcorrection. Separate operations for the lateral and vertical muscle, where the deformity is due to impairment of function in both, is certainly more exact and satisfactory than the transferred insertions or partial shortening as suggested by some. It has been my good fortune to see a number of the cases on which Dr. Banister has operated by his method, and I have had every reason to congratulate him and the patients on the excellent results he has secured. He has always urged a more careful study of the mechanics of our squint cases, as a most important basis of our operative procedure, and the results which he has been able to show more than justify his insistance on this point. I have employed his technic for muscle tucking for a number of years, and have found it to be both simple and effective.

Dr. J. M. Banister, Omaha, Nebraska (closing): Dr. Woodruff appears to think it is an accepted proposition to have the antagonist tenotomized. On the other hand, it is becoming the fashion to decry tenotomy in these cases, and I would refer the Doctor to a book just out, "Surgery of the Eye", Török and Grout, for a statement to this effect.

A good many oculists of note seem to have embraced the opinion that the antagonist in paretic conditions should not be tenotomized, and that the technical advancement of the paralyzed muscle should be the sole operative procedure. In my opinion, the tenotomy of the antagonist is an essential step.

In the case I showed upon the screen, the girl had a complete paralysis of the inferior rectus muscle and a partial paresis of the internus, resulting from a severe accident seven years before, with the fracture of orbital bones, the eye being markedly deviated upwards and outwards. The screen picture shows a perfect result as described in the paper.

Dr. Duane says advancement of the superior rectus causes diplopia in the lower visual field in cases of paralysis of the superior rectus. I do not doubt it, but my paper is not a plea for the technical advancement of the superior rectus. If one disconnects the tendon of the superior rectus and attaches it nearer to the cornea, one will almost certainly get too much upward pull, and possibly a little pull also from the muscle's not being thoroughly paralyzed, and in that way there results an exaggerated upward mechanical traction, which overmasters the inferior rectus, with diplopia in the lower visual field.

In my method, a tuck is taken in the superior rectus without attaching the muscle insertion nearer to the cornea. I never get diplopia in the lower field by the method described in my paper. I never do the technical advancement.

HISTOPATHOLOGY AND MICROSCOPY OF THE LIVING EYE.

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In the past year, several courses in the technic of using the slit lamp and corneal microscope have been given in this country. They have been well attended by American ophthalmologists, and I have been interested in the enthusiastic manner in which this branch of our work is being taken up. However, it occurred to me that many who are about to undertake this recently developed method of examination will find themselves lacking in a knowledge of histopathology that is almost essential to competency in this field. So, when I received an invitation to read a paper before this body, I felt that this would be a fitting subject to bring before you for discussion.

The Czapski binocular corneal microscope has been used in ophthalmology for many years, but it was of little clinical value with its source of illumination, that consisted of a six volt lamp mounted in a tube with a reflector. It was not until after Gullstrand presented his slit lamp at the Heidelberg Ophthalmological Congress, in 1911, that its scope of usefulness was greatly enlarged, and since that time a new field for scientific investigation has been opened, which promises to widen our knowledge of living pathology. It is possible that, with the increased illumination that we now have, the corneal microscope may soon occupy as important a place in our instrumentarium as the ophthalmoscope. If this should prove to be the case, ophthalmology can contribute much to the general science of medicine through the study of normal and abnormal living tissue.

The observations with the ophthalmoscope of changes in the blood vessels in arteriosclerosis, the nature of exudates in the hypertension of nephritis and numerous other conditions, have been a distinct contribution to our knowledge of these diseases. Prior to its introduction, the study of minute path-

ology was confined to the pathologic laboratory, where morbid tissues were sectioned, stained and examined under the microscope. Microscopic sections are thin and include only a limited portion of any diseased organ, which makes it impossible to formulate ideas of topographic alterations, unless the organ is sectioned in series and reconstructions made. This, a laborious process at best, could only be undertaken by a few specially trained workers.

The ophthalmoscope, however, opened a field that permitted all, who cared to master the technic, to study the whole fundus during the course of the disease, and watch the minute changes that took place from day to day along a whole vascular trunk, or in a wide area of the retina, choroid or optic nerve, to say nothing of changes in the media and other portions of the eye.

It has been possible in the past to examine the anterior segment of the eye only with moderate magnification, but now it can be scrutinized with the corneal microscope, aided by the slit lamp, or one of the other brilliant sources of light that are obtainable.

Now blood corpuscles are visible in the normal conjunctival vessels and their motion can be seen without difficulty. New vessels in the cornea or on the iris can be studied from day to day, and the behavior of exudates in the front part of the eye may be watched. Corneal nerves are seen in microscopic sections only when the tissues are subjected to special staining methods, but with the corneal microscope and slit lamp they can easily be demonstrated.

The methods of examination are complicated, and with our present instruments, more training and skill are required than is necessary to become proficient in the use of the ophthalmoscope.

Progressive ophthalmologists are beginning to feel the need of mastering the technic of using the corneal microscope, because it is of value in every day clinical work. With it, uveitis can be diagnosed earlier than formerly, and changes in the lens may be studied in detail. The instrument is not a scientific toy for the scientist who revels in unraveling the intricacies of a complicated structure, but it may be put to practical use by the clinician.

We, in America, have been slow in adopting the corneal microscope, and most of the pioneering has been done in

Europe. The work of Vogt, Koeppe, Koby, and a number of other Europeans, is familiar to you. In this country Bedell, Gradle, Von der Heydt and others have been doing splendid work. In the past year, many have availed themselves of the special lectures and demonstrations that have been given in this country by Mr. Basil Graves and Professor Koeppe, and knowledge of this subject is rapidly being disseminated. The demand for instruction in this branch will continue, and in the future it will be one of the subjects required in a well balanced course of graduate ophthalmology.

With this before us, we must provide for adequate instruction in morbid histopathology as an addition to the technic of microscopy of the living eye. It is true that more is being learned about the structure of the vitreous, corneal nerves, exudates in the anterior chamber, and lenticular changes, during life, than has hitherto been demonstrated in pathologic sections. However, we cannot ignore the necessity of a thorough understanding of morbid microscopic histopathology in order to interpret intelligently the pictures that we see with the corneal microscope. Inflammation in portions of the eye that are not visible with the corneal microscope, may produce secondary changes in the aqueous and vitreous that can be seen in the beam of light, so the study of histopathology should not be confined to the anterior segment, but should include those structures that might influence the transparency of media or result in other secondary changes in the front of the eye. Many things that cannot be seen with the corneal microscope are easily demonstrated in stained microscopic sections. For instance, the lamellae and fixed corneal corpuscles, as such can be beautifully shown in stained sections. The relation of Bowman's and Descemet's membranes to the substantia are better understood if they are first studied in stained microscopic sections. Without having previously seen the warts on Descemet's membrane in sections, one would be at a loss to locate them.

Before one can interpret changes in the iris, he must be familiar with its histology and anomalies. The anterior border layer contains the chromatophores, and determines its color. There are great variations in the distribution of the pigmented cells in normal eyes, and one must be familiar with the various types. This can be demonstrated best in sections of the iris, and later studied in the living eye.

Recently, I witnessed a demonstration of the layers of the crystalline lens, and heard the instructor say that the various parts were clearly visible and with a slight movement of the light the epithelium of the anterior and posterior lens capsule could be brought into view. This, of course, is impossible, because there is no epithelium on the posterior capsule of the adult lens. It is confined to the anterior capsule.

It is unnecessary to cite other examples where a familiarity with histopathology would aid in the study of the pathology of the living eye. It seems to me that there can be no argument against the necessity of combining the teaching of the two subjects. This can be done either as combined courses or dividing it into two parts.

The combined course would probably be most valuable, and could be accomplished by teaching the histopathology of the cornea in the beginning, on the same days that the mechanical and optical principles of the slit lamp and corneal microscope were being considered, and then keeping the histopathology one step in advance of the instruction on the living eye. This would necessitate lengthening the period of time for instruction, and might require two instructors instead of one.

If the subjects were divided, a systematized course in histopathology should precede the course in microscopy of the living eye. Courses in histopathology should be arranged in several of the ophthalmic centers in this country, for the express purpose of preparing men along the lines that have been suggested above. While the number of eye pathologists in this country are few, they exceed those who are qualified to teach microscopy of the living eye; and an effort should be made to stimulate their interest in arranging for such classes.

Intensive instruction for several hours each day, for a period of at least two weeks, should be given in each subject. By this method, small groups could be made familiar with microscopic pathology, which will aid greatly in the microscopic study of the living eye. Without a knowledge of histopathology, erroneous interpretations are bound to be made, and the value of the corneal microscope and slit lamp will not be appreciated.

DISCUSSION.

DR. HARRY S. GRADLE, Chicago: Dr. Finnoff's paper is one of the few papers upon slit lamp microscopy of the eye that can be actually applied to daily practice. Many of the papers in the past have dwelt upon technicalities and minute changes that are of interest to the investigator rather than to the man who is using the slit lamp in his routine office work. As a result, Dr. Finnoff's paper is very timely and should be of great benefit, as it opens up a free discussion in which those who have used the lamp may rise in meeting and give their experiences in the practical application of slit lamp microscopy.

In speaking to Dr. Bedell before the meeting, I decided to confine myself to one particular topic, and I shall speak of the conjunctiva. There is little to be gained with the slit lamp in connection with the conjunctiva, unless one resorts to vital staining; but with vital staining, a great deal of practical importance can be learned in microscopy of the living conjunctiva. Various stains may be used, and have been for a long time, but we obtain the best results with a stain recommended by Marks of Leyden a few years ago—a 5 per cent solution of rose Bengal. This gives an intense, deep red stain of a rather peculiar nature. In the first place, in a perfectly normal eye, the mucoid margin of the lid is sharp and clear cut, but if there has been recent inflammatory process in the eye, this line is jagged and interrupted, and it has seemed to me in using this stain the last year or two that in cases of burning, or irritation of the lid, such as catarrhal conjunctivitis, there is a hyperemic condition found. Another practical point is that the same symptoms occur from errors of refraction, which of course are relieved by glasses, but glasses are not indicated where the symptoms are due to localized irritation, that is, a fibrosis or localized hyperemia. With this vital staining, we have a point of differentiation as to whether or not the conjunctival condition is one of the factors in producing the symptomatology.

Vital staining of the palpebral conjunctiva is rather peculiar. The superficial cells of the conjunctiva will stain in an irregular, diffuse manner. There will be little splotches of stain on both normal and infected conjunctiva. These splotches bear no relation to each other, and are not related to the areas of most intense hyperemia. Dr. Jackson suggested this might bear out Lindner's theory of infectious conjunctivitis. Very possibly. I believe a great deal more can be learned from careful study of the conjunctiva with vital staining, and undoubtedly other stains will be found that will teach us more than we now know.

Another phase of slit lamp microscopy of the conjunctiva is observation of the small, invisible capillaries. One would expect that the capillaries at the corneal margin would be the best place for capillary microscopy, but they do not bear out the findings of capillary microscopy elsewhere. Those interested in this work have noted the peculiar condition in cases of hypertension from diabetes. In hypertension from other causes, the capillaries of the lids have a twisted appearance, while in diabetes they have a typical mushroom appearance. This does not hold true at the corneal margin, but on the other hand, the capillaries of the conjunctiva show the same changes that we will find in the vessels of the retina. You will find contortion of the capillaries, and it is almost as easy to

follow the course of the hypertension in the study of the capillaries of the conjunctiva as in the study of the retinal vessels.

Too often do we see conjunctival hemorrhages, which are of no great import except that they are extremely disfiguring. We find many of these patients have recurrent hemorrhages. There is a weak point somewhere in the vessel wall, and many times, by careful study of the vessels that lead to this hemorrhage, we are able to find the one that has a weak wall, close it by actual cautery, and prevent future recurrence of such hemorrhage.

These are practical points that may be of use in daily practice. I am not speaking of points of interest to the investigator, but to the men who are using or will use the slit lamp in daily routine practice.

DR. ARTHUR J. BEDELL, Albany, New York: You probably noted a recent editorial in the American Journal of Ophthalmology, in which it was stated, "As a routine part of the examination of each patient the use of the slit lamp is too time consuming to be justifiable." I will endeavor to show you why it should be used in a routine manner.

With the ophthalmoscope, it is easy to find a lens opacity, but without slit lamp examination, it is impossible to locate certain forms of cataract accurately. It is a decided advantage to know the detail of the structure involved in every lens opacity. These slides show the difference between traumatic cataract and those without any evident trauma.

We find an opacity in the exact center of the lens which in no way interferes with the visual capacity of the eye. Scattered irregularly throughout the lens there may be white dots. These may be more or less concentrically arranged, be in the periphery of the lens, or throughout it in regular layers. Some white dots are seen in practically every lens. Stationary and progressive cataracts can be separated. The differentiation of the lens opacities would take up more time than is allowed for this discussion.

In the beginning of my slit lamp work, I maintained that the pigment cells seen on the anterior capsule, either as isolated cells or in groups, were congenital in origin. I have seen this same type of pigment cell in cases of chronic uveitis, in which they developed during my observation of the cases. These masses and stars seem to be the result of an inflammatory process.

I believe the slit lamp has aided more in the early diagnosis of uveitis than any other single instrument of which I have knowledge. The deposits on the posterior surface of the cornea are seldom limited to a triangular form, certainly not in the early stages of the disease.

With the slit lamp, we can study the zonular fibers of the lens and trace them to the ciliary processes, the anterior capsule, the equator and the posterior capsule. In dislocation of the lens, we can tell the state of these fibers. In some cases there are no fibers adherent to the capsule, while in others there are short, bristle like hairs.

In this case of marked shrinking of the eyeball with absorption of the iris, it is possible to see the ciliary processes and follow the zonular fibers from them to the lens.

With a few slides, we will consider the subject of vitreous and vitreous prolapse. One shows some normal vitreous fibers on which there are minute pigmented deposits. Another exhibits an uncommon type,

a circle of threads with an enlargement of fibers which resemble twisted cotton strands, with here and there vitreous hemorrhages. Still another shows that some of the vitreous is filled with small, floating pigmented areas.

In closing, I wish to express my approval of Dr. Finnoff's suggestion that the study of histology and pathology be correlated with the slit lamp examination of the eye. (Thirty original lantern slides were used to illustrate the discussion, which has been much condensed.)

DR. EDMOND E. BLAAUW, Buffalo: I am one of the first to use this instrument. Most men speak of the difficulty of the technic, but it is not so difficult. The important thing is that you should begin with the lower magnifications. It is not so much the magnification as it is the illumination. It is getting a pencil of light—limited, not diffused—that is of the greatest value. If you examine with a magnification of 20, and then go on to 40 and 60 and even 100, you do not get very much more. Slit lamp illumination is of the highest value. I had been doing ophthalmology twenty-five years, but I did not know anything before I got the slit lamp.

For example, say you have a foreign body; you examine the patient and do not find anything in a routine way. If you will use your microscope, you will find sometimes that the thing is due to a very slight change in the cornea—you will find a very fine corneal defect, and I have come to the conclusion, that in these particular instances, when the patients think they have something in the eye, we are dealing with a slight infection. If you use a slit lamp the whole subject clears up.

There has been a good deal said about the difficulty in differentiating superficial corneal affections, which are often herpetic and more frequent than one would suspect. Sometimes they are in the form of fine corneal lines. I think that is the form seen by von Szily. He published a paper about linear corneal defects (Heidelberg Ophth. Ges. 1913, S. 56).

I do not know whether you agree with me that the most difficult subject in ophthalmology is the cornea. In 1898, the first part of the second edition of Graefe-Saemisch Handbook of Ophthalmology was published, but they have not yet published the part dealing with the cornea, and the cornea even with the slit lamp is difficult.

In regard to the lens, you are not able to differentiate the posterior lens capsule unless you use the slit lamp. I have heard statement after statement; for instance, Dr. Friedenwald, who before he used the slit lamp was not able to differentiate it. The slit lamp is a valuable aid in informing us about the localization of opacities.

DR. WILLIAM C. FINNOFF, Denver (closing): The purpose of the paper was simply to call attention to the fact that we are dealing with microscopic pathology when we use the slit lamp and corneal microscope; and unless we have a thorough knowledge of the histopathology of the eye, it will be of very little value. You will be disappointed in your findings unless you are thoroughly familiar with the structures of the normal and of the pathologic eye.

BLASTOMYCOSIS OF THE CORNEA, WITH A REVIEW OF REPORTED CASES OF BLASTOMYCOSIS OF THE EYE.*

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Blastomycosis, or blastomycetic dermatitis, is a chronic infective disease of the skin, which occurs as a rule in adults between the ages of twenty and sixty years. It is caused by a fungus. Beginning as a papule or nodule which frequently breaks down, there is formed a characteristic indolent wart like lesion, which later presents a raised papillomatous or verruciform appearance. This becomes marked in the chronic cases. Odd features of the disease are that the general health of the individual is undisturbed, and pain is practically absent. Blastomycosis may attack any part of the skin of the body, but has a predilection for the extremities and the face. It attacks the eyelids frequently.

In May, 1894, at a meeting of the American Dermatological Association, Gilchrist demonstrated sections from a peculiar skin disease, caused by a yeast like fungus. The case was one of scrofuloderma, in the tissues of which Gilchrist was not able to find tubercle bacilli. He found, however, numerous curious bodies throughout all the sections. They presented the appearance of parasites, and, although from lack of material there was no opportunity afforded of making cultures, for inoculation experiments, Gilchrist expressed the opinion that these bodies would be found to belong to plant rather than animal life. Further examination appeared to verify the conclusion that they might be classified as blastomycetes. Gilchrist later reported this case in detail[1]. Later, Sanfelice, Fermi and Aruch, Maffuci and Sirleo, and Tokishigi found similar lesions in animals, in which similar bodies played a pathogenic role.

Ricketts[2] made a most exhaustive study, in which he takes up:

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*From the Pathological Laboratory of the Montreal General Hospital.

1. The history and pathogenic properties of oidia.
2. Summary of socalled blastomycetic infections in man.
3. Clinical, mycologic and histologic study of twelve cases of oidiomycosis of the skin.
4. Critical analysis of blastomycetic infection of the skin.

Organisms isolated from cases of blastomycetic dermatitis seem to fall into three groups, which have so many histologic and morphologic features in common, that they probably represent different species of a common genus. They all belong to the type of fungi that Link designated as oidium. He gave this name to a genus of the fungi having an elementary organization, many species living as moulds. They have more or less ramifying and segmented filaments, which produce at their extremities short cells, very often rounded. These cells, which are united end to end, rosary like, detach themselves when mature. These are the spores, which germinate and produce filaments like those from which they came. Of the oidia, the oidium albicans was the first of medical importance, and began to occupy the attention of writers in 1840. From Robin, we learn that Berg first saw the filaments and spores of this oidium under the microscope, and recognized the disease it produced in the mouth, pharynx, esophagus, stomach and rectum. Gruby, in 1842 and 1844, published reports of his investigations, in which he considered the parasite of thrush analogous to sporotrichum. Between 1841 and 1847, many writers described thrush clinically or from a mycologic standpoint.

Robin published his classical work in 1847 to 1853. Since that time, much has been written concerning the botanic position of this fungus, and many instances of morbid processes caused by it have been reported.

To Gilchrist, however, belongs the credit of first having observed and described the skin disease in man caused by a yeast like fungus. The name blastomycetic dermatitis, which he applied, has been used by all subsequent writers. Busse, in 1894, six months after Gilchrist's brief report, published an account of a case of pyemia, caused by a pathogenic yeast. Buschke, also, reported on the same case, with special reference to the skin lesions and the organism. Among the pathologic lesions mentioned, it is interesting to see that purulent destruction of the cornea is noted. Organisms resembling oidia have been found in various conditions in man, other

than blastomycetic dermatitis and tumors. Stoewer obtained from three cases of keratitis pure cultures of a red yeast, which proved pathogenic to rabbits' eyes when injected. He also produced an inflammatory condition in rabbits' eyes with an organism isolated by Curtis. Lundsgaard obtained a pure culture of blastomyces from a case of hypopyon. Inoculations caused a haziness in the eyes of the rabbits.

Gilchrist and Stokes reported briefly a second case in July, 1896, with an extended study of it in 1898. Then followed reports by H. G. Wells in March, 1898, and by Hessler in August, 1898. Hyde, Hektoen and Bevan, reported a case in 1898 with an additional detailed report by Hektoen in 1899. Anthony and Herzog, in 1900, reported a case. W. E. Coates in February, 1900, Baldrum in January, 1900, H. W. Brayton in April, 1900. Rosenberger in August, 1900, Dyer, January, 1901, and Harris in February, 1901, all reported cases. Ricketts in his article gives details of twelve new cases, and any one interested in this subject would do well to consult this work. It is most exhaustive and complete from clinical history to histopathology.

Blastomycosis is of special interest to ophthalmologists because of the frequent involvement of the lids in the pathologic process. In reading through the case reports of most of the published cases, certainly of the earlier ones, one finds more or less obscure references to the involvement of the eyelids. No special references were made to this important feature of the disease, although certain of the illustrations show very extensive involvement of the lids and globe. In the summary of case two in Ricketts' series, the following appears: "The disease had extended over the left cheek to the nose, had surrounded the left orbit, and involved both upper and lower eyelids; the cicatricial tissue producing eversion of the lower lid." Also, in case three, "The region involved was recognized to be the left lower lid and the parts adjacent in both cheek and temple." Again, in case nine, "A small nodule appeared on the lower right eyelid, the lesion reaching the size of a bean in three weeks, when it was removed."

The importance of this disease to ophthalmologists was emphasized by Wood[6] and Wilder[7] in 1904. Wood, in his article, noted that up to that time some thirty-five cases of blastomycetic lesions of various human organs, internal and external, had been recorded, and that to that time the disease

had been observed almost exclusively in America. He gave details of nine cases in which the skin of the lids was involved.

In July, 1915, Jackson[13] recorded two cases "of a disease hitherto supposed to be rare, and still inadequately noticed in the periodic literature and text-books on ophthalmology, and to call attention to its clinical importance, the disastrous results through distortion of the lids, of failure to recognize it, and to its excellent prognosis under proper treatment."

Blastomycosis, in a large proportion of cases, has been found among laboring people, and by far a larger proportion in males than females. The age incidence varies from infancy to old age. Hessler's case was a girl affected when four to five months old. One of the cases seen at the Montreal General Hospital was a male of seventy years. The disease has been called "Chicago Disease," because most of the earlier cases were reported from that city. A number of fatal cases have been recorded. Shepherd and Rhea[12] reported one such case from the Montreal General Hospital in 1911. In quite a number of cases there is a history of injury by means of which the organisms invade the body. No part of the skin surface is immune, but the local disease seems to have a special predilection for the lids. In nine cases reported by Pusey[5], six showed lesions of the lower lid. Wood concluded one quarter of the cases had affections of the lids. The conjunctiva seems to be particularly immune from invasion, though in many cases it has suffered severely from exposure and secondary infections.

The disease begins, as a rule, as a sluggish red papule. If there has been a preceding traumatism, the wound does not heal, or only apparent healing takes place. Ordinarily, the primary papule becomes pustular in a short time, and the apex of the pustule becomes transformed into a crust, which, when removed, is seen to cover an irregular, elevated, reddened base, which secretes a small amount of mucopus. An early narrow red areola is very characteristic of the disease. Extension takes place steadily, and in two to six months the lesion will have a diameter of one to two inches. The rough granular condition of the base now becomes greatly exaggerated, and gradually a coarse papillomatous or villiform surface results, the processes of which are separated by deep irregular clefts, which are filled with pus. The appearance is sometimes

cauliflower like, the verrucose tissue rising precipitously above, and overhanging the surrounding skin. In other cases the papillomatous tissue is more flattened and shows little of the cauliflower appearance. This is always the case during the process of healing. The characteristic skin areola, red or bluish-red, rises somewhat to meet the base of the ulcer, possesses an unbroken horny layer, studded with numerous sub- or intraepithelial abscesses, which appear as minute yellowish points. If these are pricked, they yield a droplet of sticky pus in which the organisms are easily demonstrated. Sometimes the center loses its papillomatous surface and becomes depressed below the level of the surrounding tissue, either moist red granulating or dry with cicatricial healing.

In most cases, the disease progresses rapidly, with periods of comparative quiet. During the rapid extension of the disease, the whole surface is swollen, red, and secretes an offensive pus freely; the areola becomes broader, more turgescent and contains a larger number of abscesses. The pus from these is decidedly bloody. During the quiet intervals, the surface flattens and is dryer; the areola is narrower and flatter, with fewer abscesses, which now contain little or no blood. The disposition of the disease to creep gradually from the point of origin to distant areas, is well illustrated in certain cases.

Though the rough granular condition of the base, with coarse papillomatous or villiform surfaces, the processes of which are separated by deep irregular clefts filled with pus, presents a typical clinical picture, all writers emphasize the necessary differentiation from epithelioma, tuberculosis, or lupus and late syphilis of the ulcerative or warty type. To these we must add sporotrichosis.

The clinical history and appearance of many of the cases of blastomycosis are such as to indicate readily their character. Nevertheless, the diagnosis ultimately rests on the finding of the organism.

From a supposed lesion of blastomycosis, a diagnosis can often be made by a smear preparation from the purulent exudate—in the ulcer itself, or from the small superficial abscesses in the skin about the ulcer. The pus is placed upon a slide, and it is often helpful to add a few drops of a 1/10 normal solution of sodium hydroxid. This clears the exudate, and brings out sharply the double contour of the yeast bodies.

The prepared smear is first examined with the high dry lens, when the organisms will be seen in sharp outline, as double contoured, round bodies about 2-3 times the size of a pus cell. The organisms, in the majority of cases, can be demonstrated in sections made from material obtained by biopsy. In our experience, the best results have been obtained after fixation in Zenker's solution, with embedding in paraffin. The organisms stain readily with the ordinary laboratory stains, but the best one to use is Mallory's connective tissue stain. With this, the body of the organisms stains red, with the periphery a deep blue. We have seen a number of cases, evidently blastomycosis, in which the organisms were demonstrated only after many examinations or not at all. Cultivation may be easily obtained on the ordinary artificial media, such as glycerine or glucose agar, but by far the best media is "Sabouraud's" 4% glucose agar. At room temperature, the growth shows itself in 2-8 days, by a fluffy white appearance. On different media, the organisms send out mycelia, with lateral buds or coccidia.

The treatment of cutaneous blastomycosis consists in the administration of the iodid of potassium in heroic doses of from two to four hundred grains a day. By this means, the lesions can be reduced if not completely cured. X-rays also cause the disappearance of the lesions, and a favourite treatment is a combination of both. Local abscesses are, of course, treated by the ordinary surgical principles.

The case which I wish to put on record was one in which the blastomycetes were isolated from an ulcer of the cornea. C. M., a male, aged 43 years, a patient in the skin service of Dr. G. G. Campbell, was seen by me because of the involvement of his eyes in a blastomycetic infection of his face and neck, which had been present for some three years before his admission to hospital. The patient was born in Chicago, but had lived in Canada since he was four years old. In 1913, he fell from a bicycle, breaking two ribs on the left side. Abscess formation followed, proud flesh formed, and the wound discharged for some months. In 1918, while shaving, he cut a small pimple on his left cheek. Within a week, he noticed a small raised area about the size of a pea. This spread slowly at first, then quickly, involved the left lower eyelid, then went across the nose to the right lids. It then spread to the left temporal region, down under the ear to the left occipital

region, and then across, under the chin, to the right side of the neck. At my examination, it was noted that there was very extensive involvement of the lids of both sides. The right lids were completely involved in irregular, papilla like elevations, from which pus oozed upon slight pressure. The lids on the left side were practically gone. Besides a large

Figure 1. A photograph of the case mentioned in the text.

leucoma, an ulcerative process was found on the cornea of the left side.

The conjunctiva of both sides, because of the exposure, was much inflamed. As a matter of interest, scrapings were sent to the laboratory for report. Those from the conjunctiva were negative, but the slides from the corneal ulcer showed typical blastomycetes.

Snippings had previously been examined by Dr. Rhea who reported as follows: "Specimen is taken through margin of

lesion, and includes a heaped up granulation tissue with attached skin margin. One margin is heaped up with a yellowish crust, which, when removed, leaves a finely roughened tissue. This on section is pale, moist, rather soft, and is fairly sharply demarcated from the adjacent tissues. It extends under and elevates the skin margin. No definite abscesses are seen in the gross specimen. Scrapings from this tissue show many pus cells, and quite a few clearly defined pale bodies, which show a double refractile border. A few of them have a granular appearance. Most of them are rounded,

Figure 2. Generalized blastomycosis from Shepherd and Rhea's case. Giant cell with one organism in it. Stained with Mallory's connective-tissue stain. The narrow, white zone around the central, circular, darker staining area is the peripheral capsule.

though some show various stages of budding. No mycelia were found."

The corneal ulcer cleared up quickly with the ordinary measures. The patient remained many weeks in hospital, during which time his condition improved very perceptibly under large doses of potassium iodid.

Mention of corneal involvement in blastomycosis has been rare. As noted above, Buschke cites destruction of the cornea among the lesions found in his case.

Wilder[7], in his article, says "The advancing border of the disease had attacked the outer part of the right upper lid and the outer canthus. It had not quite reached the lid margin,

and the conjunctiva, although greatly exposed, was not apparently involved. The contraction of the scar tissue above and to the outer side of the lid caused an extreme ectropion, as shown in the accompanying illustration, so that it was impossible for the patient to close the lids. For this reason, the conjunctiva was edematous and suffering from a chronic inflammation. On account of the exposure of the eyeball, an infectious ulcer had started on the cornea, that threatened to destroy it, and a hypopyon half filled the anterior chamber."

In the case here reported, there had evidently been an infected ulcer of the cornea for some time, as evidenced by the large central leucoma. From the ulcerated area of the cornea, small refractile, vacuolated bodies having a double contour, often in pairs and in chains, were found.

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DISCUSSION.

DR. WILLIAM H. WILDER, Chicago: I wish to congratulate the essayist on the interesting case of blastomycosis of the cornea that he has presented, which, as far as my observation goes, would seem to be unique. It is well recognized by dermatologists, that in this disease the mucous membranes are not very frequently if ever involved, for some peculiar reason not fully understood. May it not be, in this interesting case which Dr. McKee has presented, as in the case which I reported some twenty years ago, that there was first an involvement of the cornea because of the exposure, and then a subsequent incidental infection with the blastomyces?

It is possible that there are more of these cases than is generally supposed, and that they may sometimes be passed over or confused with epithelioma, because the appearance is not unlike that or unlike lupus; and if I have anything at all to add to the consideration of this subject, it is to emphasize the importance of carefully studying the surrounding border of any ulceration around the angle of the lid or the lid margin. In this infection, there is a border such as we see in lupus or tuberculosis of the skin, elevated one-eight to one-quarter of an inch, and in and near this are found the peculiar lesions which characterize the disease, namely, small miliary abscesses, sometimes so fine that only by inspecting the lesion very carefully with a magnifying glass can they be discovered; but in the contents of these small miliary abscesses, as Dr. McKee has told you, can be found the peculiar refractile bodies, about 10 microns in diameter, which characterize the growth.

I would like to present the picture of a case which I observed and presented to the Section on Ophthalmology of the American Medical Association in 1904, in which there was also threatened destruction of the cornea, but the inference was that the corneal lesion was because of exposure and not the result of the growth of the blastomyces in that structure. The patient was a man of 60, a laborer, who had a lesion on his right upper eyelid, which began at the external canthus of the eye and spread posteriorly, involving a large area of the temple. The ultimate result was that his upper lid was drawn up. The process passed almost up to the free margin of the lid, but at no time did it get over on to the conjunctiva. Nevertheless the cornea suffered from ulceration, and it was feared he would lose the eye, but we demonstrated that it was possible, even in such a bad field, to do a successful plastic operation. A small piece of the lid was free from the scar tissue that had developed on the site of that part of the ulcer that had healed. The lid was drawn down with the usual technic of correcting a cicatricial ectropion, and a large Wolf graft taken from the arm was placed into the defect, and much to my surprise, a pretty good restoration of the lid was obtained (illustration shown).

I was able by the first operation, and then by subsequent grafting with smaller and thinner grafts, to protect his eye. The ulcer healed and a fair degree of vision was maintained. The customary treatment was carried on, the patient taking 450 grains of potassium iodid daily for a time, in conjunction with X-ray treatment, and this seemed to clear up the condition.

This picture shows a section of the growth with the characteristic miliary abscesses, and at times an appearance of the epithelium like that seen in epithelioma; but at no place do we find, as in epithelioma, the basal membrane broken, so that the condition is not subdermal, as in epithelioma, but is epidermal. As I said, it is well to emphasize this feature of the disease, and we should study the lesion very carefully to discover, if possible, the small miliary abscesses.

Enlarging one of these small miliary abscesses, we find various cellular bodies, broken down tissue, detritus, etc., and some of the peculiar coccidia.

Dr. McKee has well mentioned that we must consider the differentiation of sporotrichosis from this condition. We must have in mind the various diseases affecting the angle of the eyelids—especially lupus, epithelioma, blastomycocis and sporotrichosis, and of course the difference will be found by making cultures for the characteristic organisms, which can be readily made on any of the sweet agars.

Dr. William C. Finnoff, Denver: I will not take much of your time in a discussion of blastomycosis, because my experience in this condition is exceedingly limited and is confined to cases that occurred in the extremities, and one of the cases that was reported by Dr. Jackson.

The thing that I think needs to be emphasized, is that in these cases we must take first cultures, then smears, and make sections of the lesion, because the finding of the organism will establish the diagnosis, and the treatment is almost specific. Early diagnosis will of course prevent the unsightly deformity that you have seen in the illustrations.

ALLERGIES IN OPHTHALMOLOGY.
(Preliminary Report).

ALBERT LEMOINE, M.D.

KANSAS CITY, MO.

The question of allergies in Ophthalmology is by no means a new one. The subject received its impetus by the researches of Arthus and Uhlenhuth in 1903. Arthus[1] found that phenomena similar to allergic reactions could be produced, and Uhlenhuth[2] discovered that the protein of the lens was tissue specific and not species specific. These discoveries stimulated a great number of investigators to study further the subject of local anaphylactic phenomena in relation to diseases of the eye. The work of these investigators brought out a number of observations that may be used as a foundation upon which to build theories of allergies in certain clinical entities.

There are a few important facts upon which depend the possible allergic phenomena as met in ophthalmology, that should be mentioned. It has been shown that in an individual hypersensitive to a certain antigen, all body tissues are sensitive to that substance, and when that antigen comes in contact with any tissue an allergic reaction results. An added hypersensitivity also results at the point of inoculation, and if an intoxicating injection is given, there results a general anaphylactic shock with a local reaction at the point of each inoculation. These phenomena have been found to be especially true as concerns the eye. In other words, the eye in an individual hypersensitive to a certain antigen may develop an ocular reaction whether the antigen comes in contact with the eye or is introduced into the body, and this reaction will be more marked if the original inoculation was in the eye. In this paper, the term allergy is understood to mean a local reaction to an antigen.

With the above facts in mind, I wish to report a few cases which suggest an allergic nature of some diseases of obscure etiologic origin.

CONJUNCTIVA.

The patients in the following cases of conjunctivitis all had negative Wassermann and von Pirquet, and the smear from conjunctival secretions showed no pathogenic microorganisms.

CASE 1: T. K., male, age 6 years, presented typical clinical picture of vernal catarrh of the limbus, of three years' duration. Wright's stain of the secretions showed 20% eosinophilia. Found hypersensitive to wheat (grain), lettuce and orchard grass. There was no improvement until wheat was removed from the diet. After it was removed from diet, the condition immediately improved markedly. This patient had had asthma for 2 years. On removing wheat from the diet, the asthma also cleared up, and since then he has only had attacks when he eats wheat or has a bad cold—the latter are very mild. During the pollenating season of orchard grass, the eyes are irritable but patient does not have vernal catarrh.

CASE 2: D. S., female, age 8 years. Had marked palpebral vernal catarrh of both eyes. Second Summer. Eosinophiles in conjunctival secretion were 12%. Patient hypersensitive to timothy and ragweed. Condition did not clear up until after frost, which is the end of the pollenating season of ragweeds.

CASE 3: L. P., age 4 years. Had vernal catarrh at the limbus for 2 years, which was slightly worse in the Summer. Wright stain from conjunctival secretions showed about 60% eosinophiles. Hypersensitive to sweet vernal grass and yolk of eggs. No improvement first two weeks. Was first seen the latter part of the Summer, and on removing yolk of eggs from the diet, the condition cleared in seven or eight days. On three occasions during that Winter, the patient was given egg yolks, each time precipitating a marked recurrence of all symptoms and clinical findings, which cleared in seven or eight days after removing eggs from diet. This Spring and Summer the eyes were irritable but there was no typical vernal catarrh.

CASE 4: Male, age 8 years. Patient had a vernal catarrh at the limbus. This was his first attack. Eosinophiles 10%. Negative to all common foods and Spring pollens. Was unable to follow up this patient.

CASE 5: B. S., age 29 years. Had a palpebral vernal conjunctivitis for the past two Summers, presented himself early in June with a recurrence. Eosinophiles 17%. Very sensitive to orchard grass, timothy, sheep sorrel, sweet vernal and red top. About the first of August, got some better but did not clear up until Fall.

CASE 6: J. D., age 13 years. Had vernal catarrh at the limbus. This was his first attack. Found hypersensitive to sweet vernal, western ragweed, and grant ragweed. Did not improve until the pollenating season of those plants was over.

CASE 7: M. F., age about 30 years. Suddenly developed a very severe conjunctivitis. Eosinophilia 6%. With ordinary treatment, was getting worse; lids and bulbar conjunctiva became edematous. On inquiry, it was found the patient had changed face powder a few days before onset of present trouble. Patient was advised to discontinue its use, and the condition cleared in three or four days without any further treatment.

CASES 8 to 14: Since these cases were so similar, they will all be described together. They were patients in adult life, who had conjunctivitis that came on in the Spring or Summer. They were all hypersensitive to one or more of the pollens prevalent at that particular season, and none showed any permanent improvement until the pollenating season had passed. All other sources of irritation that might cause a conjunctivitis were excluded. They would get better for a few days, and suddenly relapse with the same condition. In many instances these relapses came on following a car ride.

In this series, there are six cases of vernal catarrah which are all the essayist has seen in the past three years. Of this number, only one was negative to the common foods and pollens for which the patient was tested. Since it was possible to follow the progress of this patient for only one week, there might have been an error in the diagnosis, or it may be that the patient was exposed and hypersensitive to some uncommon antigen for which the patient was not tested. The remaining five cases were all sensitive to one or more of the pollens. Two cases were sensitive to articles of food; namely, wheat and egg yolk. Vernal catarrh is essentially a disease of the Spring and Summer, but, as is well known, there are cases which persist, and usually in a milder form.

throughout the Winter. This is what was found in case 3, which promptly got well when wheat was removed from the diet. In case 4, the disease was almost as severe in the Winter as in the Summer. Since the condition cleared up immediately on removing egg yolk from the diet, the reaction apparently was almost entirely dependent upon hypersensitivity to egg yolk rather than to the pollen. In summing up this very small series, it seems that vernal catarrh is essentially an allergic reaction to antigens, most commonly the pollen of plants, and occasionally articles of foods. Those sensitive to articles of foods are those persisting throughout the Winter.

In case 7, the patient was sensitive to the powder she was using, but it is a question whether the patient had an idiosyncrasy to an irritating substance in the powder, or whether this was an allergic reaction.

The last six cases of conjunctivitis discussed together are similar to cases we all have frequently met, and which in the past we have been unable to explain. The fact that these patients had a condition of which we cannot determine the etiology, and the fact that they were sensitive to a pollen prevalent in our community at the time of its occurrence, and that the condition cleared up at the cessation of the pollenating season, certainly suggests an allergic origin of these conjunctivitides.

Trachoma.

Case 1: E. M., age 30. Had inflammation of the right eye 10 years ago this Summer. Does not know the diagnosis. Had another similar attack last Summer. The patient was first seen on September 25th, with a recurrence of a week's duration in both eyes. At that time, he had marked vascularity of the upper 1/3 of the right cornea, with opacities extending into the center of the cornea. Also, had advanced cicatricial contraction of the conjunctiva. The conjunctiva of the left eye also showed a marked cicatricial contraction, with a slight pannus above, with ulcers at the lower margin of the pannus that stained with fluorescin. He was found hypersensitive to ragweeds, timothy, sweet vernal, and red top. Under treatment, he was improving quite rapidly. On October 3rd, he took a ride in the country for the first time since the onset of his trouble. That night, his eyes were much worse

but gradually improved again under treatment. On October 9th, we had our first killing frost, and on October 11th the patient volunteered the information that his symptoms had practically all disappeared, and the clinical findings were much improved.

CASE 2: M. H., age 12. First seen July 31, 1923. At that time patient had marked mucopurulent discharge with photophobia both eyes. There were granules on all the lids, with a marked disturbance of the circulation in the tarsus of the upper lids. There was a slight pannus of the upper portion of both corneas. Under treatment, patient got well in October and was permitted to return to school. Returned with a recurrence in April, 1924, and again did not clear up until Fall, when she was permitted to return to school. Patient was not seen again until July 1925, when there was a marked recurrence of symptoms and clinical findings. At this time, the right eye presented cicatricial contractions of an old trachoma. The left eye had many follicles with slight contraction. Patient was tested for pollens and found hypersensitive to ragweeds, sweet vernal grass, timothy, orchard grass, and slightly sensitive to June grass and sheep sorrel. Patient had shown very little improvement under treatment. First killing frost was on October 9th. Two days later, patient had improved 50%, and was nearly well four days after the frost.

CASE 3: B. T., age 16. First saw patient in the Summer of 1923. Had been treated for trachoma off and on for the past five years. At that time, both eyes presented the picture of old trachoma. There was also a pannus of both eyes, the right extending to the center of the cornea, the left about 3 mm. into the cornea. Treated until Fall, when she was markedly improved. Patient was worse the following Summer, again having a recurrence of the pannus. In October, was well enough to return to school. Returned in the Spring of 1925 with an exacerbation. All through the Summer she had a pannus of the right eye, which would not clear under treatment. Tested for pollen and found to be hypersensitive to wheat (globulin), sweet vernal, timothy, June grass, orchard grass, yellow dock, and ragweeds. Patient was last seen October 17, and was nearly well clinically and free from symptoms.

CASE 4: C. C., age 52. Came in this Fall with a recurrent attack of sore eyes. Has had similar attacks for the past

eight years. Always worse in the Summer. The past two Summers it came on in August, as soon as he began cutting corn fodder, and cleared up latter part of the Fall. This year, the present attack came on as soon as he began cutting corn fodder early in August. Thinks his eye trouble is due to dirt in corn. Tested to pollens and found very sensitive to all ragweeds, sweet vernal, yellow dock, and orchard grass, but not hypersensitive to pollen of corn. Patient presented an old trachoma in the cicatricial stage, with an active pannus of both eyes. Was last seen on October 15, and was very markedly improved.

The etiology of trachoma, a disease that has baffled ophthalmologists from time immemorial, still remains an unsolved problem. Fortunately, or unfortunately, in the past year I have had but few cases that I felt positive were true trachomas. The 4 cases above reported are the only ones in which I had an opportunity to make the tests for hypersensitivity to antigens, since the beginning of this work. Such a small number of cases, and all of them having a history of recurrences at a certain season, which does not coincide with the history of the majority of trachoma patients, makes this a very poor series to draw conclusions from, but the fact that they are all hypersensitive to several pollens prevalent during the season of their recurrent attacks, certainly is suggestive that some of these cases we have been classifying as true trachoma might be allergic in origin. The patient in case 2 volunteered the information that his eyes always got worse when he began to cut corn fodder. Since this patient was not hypersensitive to corn pollen, there must have been some irritating substance at this season that caused his exacerbations. He was sensitive to all the ragweed pollens, and they frequently are found in corn fields. In the near future, I hope to be able to draw definite conclusions that will tend either to prove or disprove this hypothesis.

PHLYCTENULAR KERATITIS.

Name	Age	Von Pirquet Reaction	Wassermann Reaction	Hypersensitive to	Involvement	Remarks
Case 1 O. M.	21 mo.	+ +	Negative	Negative	Both corneae	Got well on tuberculin treatment. No recurrence in 2 years.
Case 2 L. M.	6 yrs.	+ +	Negative	Negative	Both corneae	Two attacks. No more since tuberculin 1 year ago.
Case 3 L. B.	2 yrs.	+ +	Negative	Not made	Both corneae	Two attacks. No more after tuberculin treament 1 yr. ago.
Case 4 E. C.	3 yrs.	+ +	Negative	Negative	Cornea O. S.	Two attacks. None since tuberculin treatment 2 years ago. Had Pott's disease.
Case 5 H. A.	5 yrs.	Von Pirquet and Manteau negative	Negative	Strawberries	Both corneae	Cleared in a few days after removing strawberries. One relapse from eating strawberries.

DENDRITIC KERATITIS.

CASE 6: Mrs. J. D., age 52 years. First seen April, 1923. Had corneal ulcer left eye; no pathogenic microorganisms found. Ulcer was very indolent and lasted three weeks. Had a recurrence in December of the same year, which lasted a week. Had a second recurrence in August, 1924, which persisted for nearly two weeks. Another recurrence in February 1925. The ulcer at this time was a typical dendritic ulcer in the lower quadrant, about 1 cm. in length. In going over her history, it was found that she had eaten a five pound box of chocolates in the two days preceding the onset of her ulcer, and since the onset was eating very little else but chocolate candy. In going back over the history of her previous attack, she associated each one with excessive eating of chocolates. She also volunteered the information, that each time she ate several chocolates, her nose became red and puffy, which she

termed "a chocolate nose." She was found to be quite sensitive to chocolate, and on removing this from her diet, she got well in two days. She was advised not to eat any more chocolates, and has had no recurrence since.

CASE 7: Mrs. M. W., age 63. First seen in April, 1923. Had recurring dendritic ulcers all Summer until the 1st of September, when she had no more ulcers until April, 1924. In that summer, the ulcer healed quite readily and no more occurred the remainder of the summer. Returned August 2, 1925, with another dendritic ulcer of the right cornea, which remained irritable until October 3rd. During this attack, she was tested for hypersensitivity to pollens, and was found to be sensitive to sweet vernal grass, sheep sorrel, June grass, giant ragweed, orchard grass and timothy.

Of the five cases of phlyctenular keratitis, there were four which were hypersensitive to tuberculin. This makes 80%.

The other case was hypersensitive to strawberries, the condition clearing immediately on removing these from her diet. A later recurrence from eating strawberries promptly cleared when they were discontinued. As pointed out by Wood[3], there has been sufficient experimental and clinical evidence to warrant the assumption of an allergic reaction to tuberculoprotein in cases that are hypersensitive to tuberculin. The number found hypersensitive to tuberculin in 942 cases, collected from the literature by Gibson[4], was 85%. In my small series of cases I got approximately the same, 80%, and the remaining 20% is made up with a case that was hypersensitive to strawberries. Summing up these two factors into allergic reaction, gives us the most plausible theory of the etiology of phlyctenular keratitis yet advanced.

The dendritic keratatis in case 6 seems to be definitely associated with hypersensitivity to chocolate. I attempted to get the patient to eat a lot of chocolate, to see if it could be produced again, but the patient was so firmly convinced it was the cause of her eye trouble, that she refused to do so. The history and the findings in case 7 is at least suggestive of an allergic origin. Two cases is an insufficient number from which to draw conclusions, and I do not wish to be understood that I think all cases of dendritic keratitis are due to allergic reactions, but I feel that we can safely assume that at least some cases are allergic in origin.

Parenchymatous keratitis was produced by experimental anaphylaxis by Wessely[5], and other investigators confirmed his findings. Allen Wood[3] reported a clinical case due to the pollen of corn, that cleared up when the patient was desensitized with corn pollen. I had hoped to report on experimental work which tends to show that the majority of cases of parenchymatous keratitis were allergic reactions to either treponemata pallida, tubercle bacillus or other antigens, but unfortunately I am not prepared to report on this work.

Treatment.

Having determined that an eye condition is due to certain antigens does not completely solve the problem confronting us. Why should many of these individuals become hypersensitive to the protein in question? From my observations of the cases reported, I feel that the internal secretions certainly must play an important part. One need only reflect on the marked action of adrenalin in cases of anaphylaxis, to realize the importance of internal secretions in such cases, and an allergic reaction is nothing more than a local anaphylaxis. Since adrenalin acts favorably on anaphylaxis, there must first be a disturbance of the adrenal function and a consequent broken balance of the whole mechanism of internal secretion. Likewise, focal infection must play an important part. The modus operandi of focal infection in these cases is somewhat problematic. It may be brought about either by sensitizing the patient by a nonspecific protein of a bacteria or its product, or by disturbing the internal secretion with a resulting increased susceptibility to protein sensitization; the patient then being exposed to the antigen in the proper dosage becomes hypersensitive. Or some patients may have inherited a susceptibility to certain proteins. Therefore, in treating these cases, it is very important to make a complete study of the patient to determine these points, and to correct any such existing exciting factor. Then, one must proceed to eliminate the exciting antigen or antigens. If this is impossible, an effort should then be made to immunize against the substance in question. Calcium lactat in doses of 10 to 20 grains acts favorably on these allergic patients.

Conclusions.

Vernal catarrh is an allergic reaction to pollens prevalent in the Spring and Summer, and to antigens either coming in contact with the eye or taken into the body as food.

Many of the irritative conjunctivitides, in which all other causes have been eliminated, are likely to be allergic reactions to pollens or foods.

It seems probable that some cases now classified as true trachomas might be allergic reactions to pollens or other antigens.

Phlyctenular keratitis is most likely an allergic reaction to tuberculoprotein and other antigens. In fact, this is the only theory yet advanced that can account for all cases of the disease.

Some cases of dendritic keratitis apparently are allergic phenomena in the cornea, due to ingested foods or to pollens.

I wish to take this opportunity to thank Dr. Chas. Eldridge for his cooperation in surpervising the protein tests on the cases at the Children's Mercy Hospital. I also wish to express my appreciation to Dr. Joseph Lichtenberg for corrections and suggestions.

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DISCUSSION.

DR. GEORGE F. SUKER, Chicago: I think the Doctor has opened up a very large topic for study, of which very little is yet known that is positive. Theory is very good—we cannot establish facts unless we have a sound theory to build upon. To put this entire question into a nutshell, you must get a substance that will answer every purpose in every instance—you must consider the physiologic, biochemic metabolism of the individual as a whole, and unless you have something that will counterbalance that abnormal physiologic, biochemic, metabolic action, you will not have any success in establishing immunity. After all, what we are after is to get something that will immunize these patients, not only from time to time, but permanently. If it were possible for these various experimentalists to get a something that would answer the same purpose as does vaccine for smallpox, there would be very little difficulty. This idea they must carry in their mind, otherwise they will not succeed in establishing that which they would like to—a cure for these patients. It is not the simple factor, but the multiplicity of the conditions. These patients are sensitive, not to one, but several things. What is the condition of this particular cell in the human economy at the time of its sensitiveness? What is its relation, its chemical reaction, and what relation does it bear to the chemical reaction of that cell when it is not sensitive? In other words, can there not be something going on in these patients who have allergic conditions in the same way that we have certain reactions—the Wassermann reaction in the spinal fluid findings of a patient who is syphilitic? It is no doubt foreign protein therapy, but the thing that does not quite appeal to me is its multiplicity, that these various patients that have supposed allergic conditions are not only sensitive to one, but to several. Then there must be some way of deciding whether there is a relation between these foreign protein substances to which they are sensitive. In other words, it is bound to resolve itself more or less into a kind of composite therapy. I think if all these experimentalists would bear that in mind, it would be better for them to endeavor if possible to get the chemical reaction, the chemical constituency, the combinations of these various irritating substances. You can have an allergic reaction in any of the internal or the external organs, but is there any similarity between them? It seems to me, as I have been looking at it in the cases I have found, there is always more or less of an element of angioneurotic edema connected with it. That is necessarily a peripheral affair; we know it is toxic; we know adrenalin is very efficacious in this condition, and these patients are improved by a certain amount of internal gland therapy. What is going on in that system? Can anything produced by the experimentalist in the laboratory establish in that individual an acquired immunity? The majority of us have a natural immunity, but physiologists have not yet analyzed immunity. We know its action, but what is it? Neither do we know what acquired immunity is. You have to take into consideration the before and after effects. You cannot get away from the allergic principle in considering the action of the ductless glands.

I was very much interested in the fact that the Doctor broached the subject of trachoma as being an allergic condition. That to my mind is an open question. No doubt there is something to it, as these patients are very sensitive. Years ago, we used to get them in Ann Arbor, hundreds

of them, sent from the northern peninsula and they were always better in the Winter than in Summer. At that time we did not know there was any relationship existing. I really believe the Doctor has struck the right idea, and I trust he will go on with his experiments.

As far as the question of parenchymatous keratitis is concerned, I wish the doctor would carry this idea—that there is just as much difference, from a clinical standpoint, between interstitial keratitis and parenchymatous keratitis as there is between interstitial nephrits and parenchymatous nephritis.

I do not wish to detract from the experiments, but nevertheless the physiologic, biologic metabolism and the relation of cell economy to the economy of the cell when irritated by these pollens must be taken into consideration.

Dr. Lee M. Francis, Buffalo: Last Summer I saw a healthy girl of eighteen with persistent bilateral attacks of herpes of the cornea, extending over several weeks, which did not respond to treatment. After careful study, no cause was found, but after typing out to foreign proteins, she was found sensitive to chocolate and corn. Upon withdrawal of chocolate and corn, her herpes promptly healed. Several weeks, later, she ate chocolate, with a recurrence of the herpes on one side. This year, however, she ate corn and a little chocolate without producing herpes.

Dr. Daniel W. White, Tulsa, Oklahoma: As you all know, trachoma is a very chronic eye disease, and you may have a pannus in a patient at 19 years of age, or at 28 years of age. In the analysis of many thousands of cases, our experience has been to have found pannus at 28 years of age. You may have a very virulent trachoma of the eyelids, or a pannus of the eyeball at this time. I believe in allergy the treatment should be started during different stages of the trachoma to secure the best results.

Dr. Lemoine speaks of the recurrence of the pannus. In our experience, we find the pannus may appear infrequently fifteen years after every form of treatment, that is, when the lids are supposed to be well. Here I may mention a boy, 13 years of age, who had treatment for three years and had been pronounced cured. Fifteen years later, he appeared at our office with a returning pannus. We found by removing the tarsal cartilage and the conjunctiva, and treating the lower lids with mild astringents, especially copper, the pannus cleared up. We also paid particular attention to the treatment of the tear duct in those severe cases. As for pannus cases, I would suggest that Dr. Lemoine should try to use his treatment as early as possible in the disease, and if he wants statistics on the treatment, I believe he will find that he will have these cases (pannus) mostly between 25 and 30 years of age. In other words, you can have trachoma for 15 or 20 years and have no pannus, and then you may have marked pannus in cases between 28 and 35 years of age.

I am glad Dr. Lemoine expressed himself on allergy. I agree with him that in summer the pannus is much worse, and have no doubt in my mind that the pollen from grasses increases the pannus and in some cases causes it.

Dr. Albert N. Lemoine, Kansas City, Mo. (closing): What I wanted was suggestions and help to complete this work. This is only a preliminary report, and I hope to have some more definite conclusions in another year or two.

INFILTRATION ANESTHESIA AND NERVE BLOCKING IN OPHTHALMIC SURGERY.

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American ophthalmologists may well be proud of the fact that the man who discovered local anesthesia, one of the greatest achievements of surgery, is an ophthalmic surgeon who has practiced most of his life in New York. It might be expected that progress in local anesthesia in this country would be such, that we could point with pride to our accomplishments in that field. This is far from being the case. Not only have we done little in leadership, we have not even been quick to follow the advances made by others. Witness the reports of the Committee on Local Anesthesia in Ophthalmic Work of the Ophthalmic Section of the A. M. A., 1921 to 1924. For example, in their last report (Trans. Sect. on Oph. A. M. A. 1924, p. 305) they say: "Notwithstanding its many advantages in many cases, it is surprising to learn that nearly a hundred ophthalmologists of considerable experience have never used infiltration anesthesia at all. Of more than 300 who use it, only about 200 use it to any extent, and among the latter only a limited number use it extensively in a variety of conditions."

Apparently the members of the Committee are not among this limited number who use it extensively, for they appear to base their report on the statements of others, rather than on personal experience. This impersonal attitude was doubtless deliberately chosen by the Committee. Per contra, the present writer will make this a statement of personal experience.

My first enucleation under local anesthesia was done in 1891. The first ophthalmic paper I wrote was "The Local Anesthetics Used in the Eye," in 1898. This explains why my attention has been directed toward the subject of local anesthesia ever since.

Most ophthalmic operations can be done with local

anesthesia. Not enough attention is paid to the anesthesia by most operators: many surgeons seem to think that if they do not cause much pain by their cutting and other manipulations, they have done all that can be expected. They seem to think that a patient should expect some pain and should not complain if he is hurt a little, for an operation is not a pleasure party. There are two reasons why this attitude is wrong. We should aim to "rob surgery of its terrors," not only for humanitarian reasons, but because in many of our operations the greatest peril comes from sudden movements by the patient; and these are caused nine times out of ten by the patient feeling something. It may not have been anything really painful; perhaps it was only something unexpected. Again, in other operations, for example on the muscles or lids, if the patient is showing distress, and is groaning or complaining and is restless, we are likely to give less scrupulous attention to details, and to do a less thorough operation, to be satisfied with less perfect technic, if only we can hurry through without hurting too much. In my opinion it is hard to exaggerate the importance of good anesthesia. It cannot be obtained without taking a good deal of pains.

Local anesthesia, skillfully carried out, will abolish pain entirely, but this is not enough. A large part of the suffering is mental, due to apprehension and fear. If the mental state of the patient is not controlled, the anesthesia is by no means well done. There are two means to be used to secure the desired mental state: (1) The way the surgeon talks about the operation from the time he first broaches the subject should be such as to inspire confidence. If the patient does not have implicit faith in the surgeon, he will behave less well, and his lack of confidence will react on the surgeon, who will not do his best work if he detects a subtle distrust in the attitude of the patient. Not only must the surgeon attend to his own direct influence on the patient in inspiring confidence, he must see to it that the environment of the patient is favorable. Pessimistic relatives, friends, and neighbors may impart by a word, a question, a look, distrust and apprehension of the most depressing and harmful character. Cheerful, hopeful, optimistic nurses and assistants are among the most important factors in securing the proper mental state of the patient.

(2) Beginning one or more days before the operation, not later than the day the patient enters the hospital, admin-

ister such sedatives and hypnotics as will secure good sleep every night, and a gradually increasing drowsiness from day to day, so that when the time comes for the operation, he will surprise everyone, especially himself, by his freedom from anxiety and nervousness. Do not be deceived by the apparent calmness and steadiness of the self controlled person who manifests no distress and assures you that he is not nervous, for example, the husky athlete; it is ten to one that he is nervous inside, and his pulse may show it already. He may be lying awake at night, picturing to himself the possible mishaps that may occur. Take no chances; give him some sedatives, sufficient to ensure a good night's sleep.

On the day of the operation give another dose early in the day, and repeat it as often as necessary, so that when the hour for operating arrives, he will be so drowsy that he pays little heed to what is going on around him, though he can be easily aroused to answer questions and obey orders. Of course, hubbub and confusion, with frequent orders and questions and evidences of inefficiency in the patient's room or in the operating room, in his hearing, are *most objectionable*.

What drugs shall we use? There are several well tried and effective drugs, and one is apt to have his own favorites, which may not be better than many others, but which he knows about because he has tried them often. Bromid in doses of ten to twenty grains three times a day, with chloral added as the day of the operation approaches, in doses of five to ten grains, will secure a drowsy, placid state; but the mistake is often made of giving the bromid in too small doses. I have used these drugs, but they are disagreeable to take, and chloral not rarely causes nausea, so I more often use barbital or barbital sodium in doses of five to ten grains, two or three times a day. I also use adaline (a bromid compound) in five to ten grain doses, two or three times a day; and may use both in some cases. An hour before the operation, a hypodermic of morphin 1/6 to 1/3 of a grain, and hyoscin 1/120 to 1/150, is given. If the patient is not sufficiently drowsy and placid when it is time to bring him to the operating room, give another dose of morphin, 1/8 of a grain more or less, and hyoscin, 1/150-1/200 gr. The advice of the Committee of the A. M. A. is to use morphin 1/6 and scopolamin 1/300 as sufficient to put the patient in a pleasant and obedient mood. In my experience, this is usually quite inadequate even when

barbital has been given. Most adults need two or three times as much. If the operation is a cataract extraction, I omit the morphin, because it causes nausea in some people. In these cases I give a larger amount of barbital, sufficient to produce drowsiness. I do not aim to produce the typical "twilight sleep," which is a much deeper state of narcosis, because it is so dangerous. The doses given above are safe and usually most efficient.

Now and then one meets a patient who has an idiosyncrasy against barbital. In these patients, a sensation of muscular weakness is produced. They "feel as if they had had a fit of sickness." If the administration of the drug is begun a day or two before the operation, one finds out this effect and switches to another drug. For a similar reason, it is wise to give hyoscin in divided doses, first giving only 1/150 gr. (less in children or very old or feeble patients) and observing its effect. If we have to do with a case of idiosyncrasy, this dose will show it. The symptoms to watch for are the pulse, which should not be too rapid, and especially the mental signs suggestive of delirium, viz., plucking at the bed clothes, talking and acting queerly.

A common mistake is to give the sedatives and fail to follow them up with a quiet environment. The patient should be in bed and in a quiet room, or, if in a ward, a screen should be placed around the bed and every effort made to favor sleep, or at least a composed and placid state. To arouse the patient by shifting him to a different bed, or changing his garments, or giving an enema or otherwise violating his peace just before taking him to the operating room is very ill advised.

The contrast in the behavior of a patient operated under local anesthesia, when in this sleepy and placid state, and of a patient with no sedatives, or only insufficient doses, is most marked in two ways. The patient is spared much distress, and he behaves vastly better at critical moments, for example during the iridectomy or the delivery of the lens in a cataract extraction.

Technic of Local Anesthesia.

Cocain is the best local anesthetic to drop in the conjunctival sac: 4% is a good strength. The solution should be sterile and preferably freshly prepared. A tablet dropped into

boiling water, and the solution quickly cooled, makes a sufficiently sterile solution, which is not decomposed enough by the brief heating to make it unsatisfactory to use. Or the solution prepared by the pharmacist may be boiled in the operating room for a few seconds, and then cooled. I like to have a small quantity of some antiseptic added to the solution, for example, chloretone, one to one thousand.

A good way to apply the cocain is as follows: Instil a small fraction of a drop and close the eye. This smarts a little, hence the small drop, since a large quantity smarts much more; also, the smarting is less if the eye is closed than if it is winked several times. After one minute, instil a larger quantity. This will not smart much, because the initial small drop has caused some anesthesia already. If there is not a well marked ischemia after this, instil adrenalin or its equivalent, and wait until the ischemia shows. Then drop in some more cocain, and repeat it every three minutes until four or five instillations in all have been made. Some patients react to the instillation of the cocain or other solution by an instantaneous spasm of the orbicularis, which expels most of the solution before it has had time to find its way all over the conjunctival sac. It is not necessary to combat this tendency at the first instillation; but after the second drop has been given, there is less discomfort caused by the subsequent instillations, and pains should be taken to have the lids relaxed and, if necessary, held by the one who is giving the drops, to favor the spread of the solution to all parts of the conjunctival sac before the patient is permitted to close the lids tightly.

The cornea and conjunctiva will be found to be satisfactorily anesthetised; but if the operation involves the iris or the ocular muscles or other deeper tissues, a different technic is necessary for the best results.

Anesthesia with Subconjunctival Injection

Begin as described above with a small drop of cocain, followed by adrenalin, and then by cocain, three or four drops. In three minutes, repeat twice. After two or three minutes, more, the conjunctiva will be sufficiently anesthetised to make possible the introduction of the speculum and the subconjunctival injection of whatever solution is chosen. For cataract and glaucoma operations, where only four or five drops of solution are needed for the subconjunctival injection, it is safe to use cocain, and,

since that is the most efficient, I prefer it. I use 1% or 2% cocain, with a few drops of adrenalin added. The conjunctiva is grasped with fixation forceps, raising a fold (Fig. 1). Into this fold the fine, sharp needle is thrust, and about four or five drops injected, making a bleb nearly the size of the cornea (Fig. 2). The speculum is withdrawn, and the eye closed. A little massage to spread the solution into the tissues where we wish it to act is advantageous, but prolonged massage with a view to favoring absorption is to be avoided, since our aim is to secure the maximum of local action and the minimum of systemic action. The ischemia produced by the adrenalin greatly favors

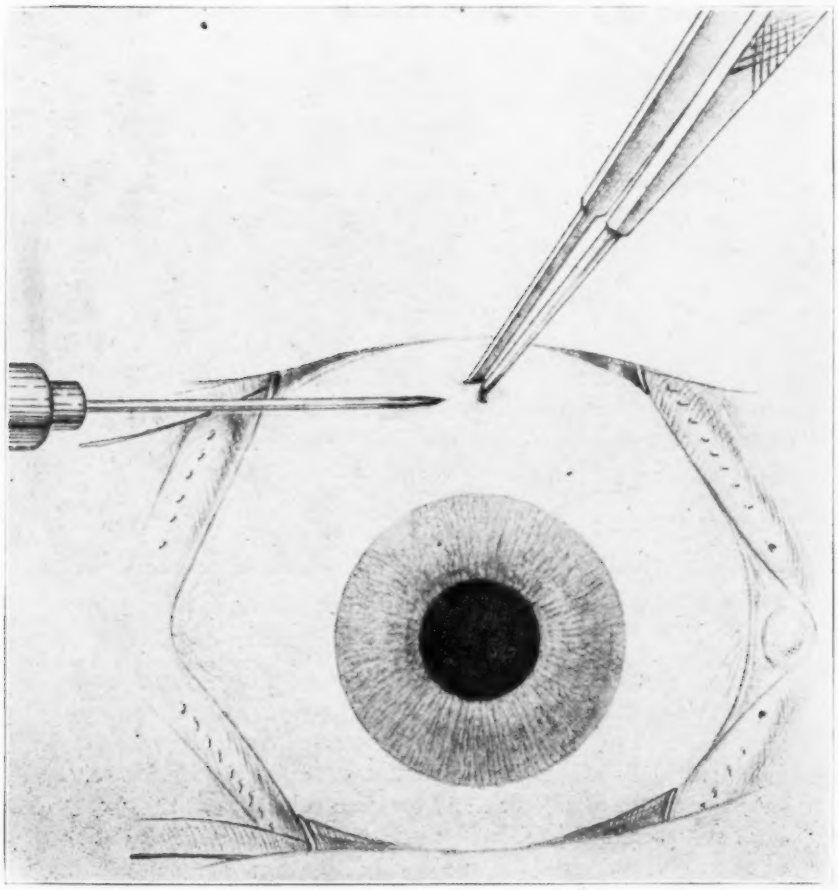

Figure 1. Method of injecting cocain for cataract and glaucoma.

this restriction of the action to the local effect, and so increases the anesthesia.

If a large bleb is made above the cornea, it may be embarrassing to the surgeon in making his section just where he wants it, and in forming his conjunctival flap. Hence the bleb should be dispersed by massage immediately after injecting the cocain, or else the injection should be made below and spread around the cornea by massage.

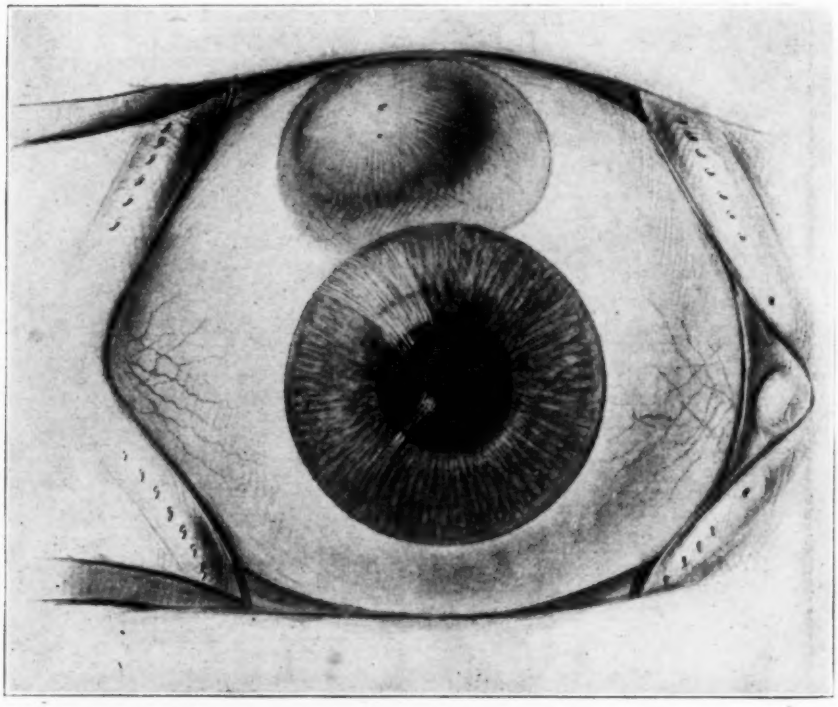

Figure 2. Appearance of bleb after a sufficient quantity has been injected for most operations.

If for some reason it is desired to have a bleb, for example to favor the cutting of a large conjunctival flap, and the solution which was injected has so dispersed as to leave an insufficient bleb, it is a simple matter to inject a little air so as to make a bleb of any size and location desired.

Then comes the tedious waiting. It is necessary to allow at least five minutes for the cocain to reach the nerves and combine with them. To inject the solution and then start

at once with the operation, is a serious fault. During this time, it is wise to flood the conjunctiva with mercurochrome or argyrol, allowing it to act for five minutes and then washing out the sac. After flushing out the sac, there will be some hyperemia, therefore drop in one or two drops of adrenalin and follow in a minute with several drops of 4% cocain, since the superficial anesthesia has had time to decrease, and reliance should not be placed solely on the subconjunctival injection.

If the operation is on the ocular muscles, a larger amount of solution must be injected; and I am afraid to use cocain even in ½ or ¼%, and so use procain in 1 to 2%, according to the amount needed: 10 c.c. can be used with safety, especially if a few drops of adrenalin are added to cause ischemia, so as to retard the absorption. The danger of serious toxic symptoms from cocain, procain, and the like, depends largely on the speed with which they enter the circulation. A very much smaller dose will be fatal when injected intravenously than when injected subcutaneously, and an injection in the limbs is less dangerous than one about the face. If barbital has been given, the danger that cocain or procain will cause cerebral symptoms is distinctly less.

In cataract and other operations, it is sometimes wise to take further precautions to secure complete anesthesia, by injecting deeply into the orbit enough procain and adrenalin to absolutely assure complete loss of sensation. In doing this, it makes an important difference just where the injection is placed. It is a common practice to inject it along the external rectus, thrusting the needle in to a depth of about 25 mm., so as to be near the ciliary ganglion. This produces a very complete anesthesia, but it also produces a paralysis more or less complete of the external rectus muscle, so that the eyeball is rotated toward the nose. This is a very awkward position for the eyeball to assume. Hence the injection should be made at the upper inner angle, thrusting the needle backward into the orbit, and injecting a portion of the solution along the internal rectus and some along the superior. This will cause the eyeball to rotate down and out and so make it easier for the operation.

Ten years ago I did a series of cataract operations with deep injections into the orbit in every case. I gave it up because I found it unnecessary as a routine, and have reserved it

for special cases. For example, if I wish to take extra precautions against squeezing, because the patient shows signs suggestive of danger, I inject deeply in two or more places enough procain to produce paresis of the recti muscles. Or if the eye is for any reason hypersensitive and I fear the usual application of cocain may not produce adequate anesthesia, I inject above or up and in, at a depth of 30 mm., about 2 c. c. of 1 to 2% procain. This causes some exophthalmos, and is made to spread about in the orbit by massage of the eyeball, so as to insure contact of the procain with the ciliary nerves, both posterior and anterior. The exophthalmos is sometimes an advantage in deep set eyes, because it makes the eye more accessible. Added to this is the advantage of greater immobility from paresis of the recti muscles. If the exophthalmos is so great as to cause undue tension on the recti muscles, thus increasing intraocular pressure and danger of prolapse of vitreous, the exophthalmos should be reduced by massage. This is very rarely necessary, because, as the procain is always injected slowly, one can see when the tissues have become so filled as to begin to push the eyeball forward, and should stop injecting. I have never had a hemorrhage into the orbit from the use of the needle in deep injections. I use a fine, sharp needle, and I withdraw it slightly or push it forward slightly in a new direction, injecting a little in each place. Occasionally, I inject the greater part in one spot rather rapidly, and perhaps it makes no great difference.

For muscle operations, the subconjunctival injection should be made deeply—under the capsule of Tenon, subcapsular, not subconjunctival. If it is made just under the conjunctiva, the resulting edema obscures the operative field and makes the operation more difficult. If the injection is made deeply along the muscle, the needle being thrust in 15 to 25 mm., not enough edema is caused to give any trouble, and the muscle is made so anesthetic that pulling on it with the muscle hook does not hurt. The quantity required for a muscle operation is at least one c. c. and it is better to use more. Frequently the anesthesia is unsatisfactory because not enough was injected, and it was not put in the right place. Rest assured that unsatisfactory anesthesia is the fault of the surgeon.

Does the local anesthetic have any injurious effect on the healing process? Undoubtedly it does. Too prolonged and

liberal application of cocain and of adrenalin have a toxic effect on the cornea. The cornea is said to become tougher and harder to cut, and of course the epithelium often desquamates, and the corneal tissue is made less resistant to harmful influences.

The only harmful effect from subconjunctival injections of local anesthetics which I have observed, has been a tendency to favor bleb formation, in doing cataract extraction with very large conjunctival flaps, when the injection was made above. Hence it is better to make a small conjunctival flap, or else inject the cocain below the cornea and spread it around by massage.

For such operations as removal of foreign body from the interior of the eye, repair of penetrating wound of the eye, perhaps with prolapse, iridectomy or sclerotomy in acute glaucoma, it is necessary to infiltrate the orbital tissue back of the capsule of Tenon pretty completely with the anesthetic solution. As the insertion of the needle, particularly if it is not very sharp and rather fine, requires some force and so is likely to cause pain at the very beginning of the operation, thus forfeiting the confidence of the patient, it is worth while to devote special pains and a little more time to producing a localized anesthesia where the deep injection is to be made. To accomplish this, first instil cocain and adrenalin in the conjunctival sac several times. Then, with the smallest sharpest needle, make a subconjunctival, superficial injection of procain and adrenalin at the site of the deep injections. Wait a few minutes for this to take effect, then insert the longer needle with gradually increasing pressure and some boring motion, until the capsule is pierced, after which the needle can be pushed in with little force as far as you wish. Do not select as the point of entrance the place where the capsule of Tenon is thickest and toughest, viz., the external and internal lateral ligaments. Sometimes it is easier to insert the needle through the skin and lid at the margin of the orbit instead of through the conjunctiva. In acute glaucoma, the deep injection of procain and adrenalin will reduce the tension very materially in many cases, if time is given for it to act, so that there is this advantage of local over general anesthesia in dealing with this difficult situation. Should the anesthesia be inadequate for any reason, the deep injection will have done some good and can be supplemented by some general anesthetic.

For enucleation, more of the anesthetic solution may be used, because some escapes when the eyeball is removed and the orbit washed out with sterile saline.

My practice is as follows: Preliminary preparation with barbital day before and 2 hours before, morphin and hyoscin 1½ hrs. before and ½ hr. before. Cocain and adrenalin in the conjunctival sac 3 or 4 times. Wash out the sac and instill 1% mercurochrome. Insert speculum and inject ½ c. c. of 1% procain containing adrenalin, 1:20000, over each of the recti muscles except the superior rectus, where 1 c. c. is injected, because there are nerves entering the orbit above which are not reached by the deep injection, and the patient often notices pain when the surgeon works on the superior rectus. One can wait a few minutes if the eye is tender or the patient hypersensitive before continuing with the deep injections, which are more painful because it takes more force to push the needle through the capsule of Tenon. Then inject one c. c. along the internal rectus and 2 c. c. deep along the external rectus. Introduce the needle just below the external check ligament and just above the internal check ligament, because less force is required to penetrate. Be careful not to stick the needle into the globe or into the periosteum. Remember that the outer wall of the orbit goes back at an angle of 45°, while the inner wall goes straight back.

Let the needle enter at least 25 mm., so that the ciliary ganglion will be bathed in the solution and all the ciliary nerves will be reached. At least 5 c. c. will have been injected by this time, and there should be a chemotic swelling of the conjunctiva all around the cornea, and the eyeball should be in a state of exophthalmos. Use a little massage to insure the spread of the solution to all parts of the orbit, but not enough to expel the solution from the orbit. Wash out any solution of cocain or procain that may remain free in the conjunctival sac before flooding with 1% mercurochrome, which is incompatible with the cocain, procain and other alkaloid solutions. Ten minutes should be allowed for the procain to combine with the nerves and produce good anesthesia. The last one or two minutes can be used to irrigate the conjunctival sac, washing out the mercurochrome and any mucus or debris. In my experience, there is not only no need of waiting 30 minutes before beginning, but there is a distinct disadvantage. From 10 to 20 is the best time, and if you wait longer than 10 minutes,

it is advisable to instill cocain a few times for the superficial effect which will have worn off by this time. Apparently it is not generally known that one can enucleate under local anesthesia eyes that are inflamed and even in a state of panophthalmitis with chemosis.

Extirpation of the lacrimal sac can be done painlessly under local in most cases, but it is much more difficult to secure satisfactory anesthesia than in operations on the globe, on account of the anatomic conditions. (See Figs. 3 and 4).

Begin by infiltrating the skin along the line of the proposed incision. Then extend the infiltration by blocking the infra-

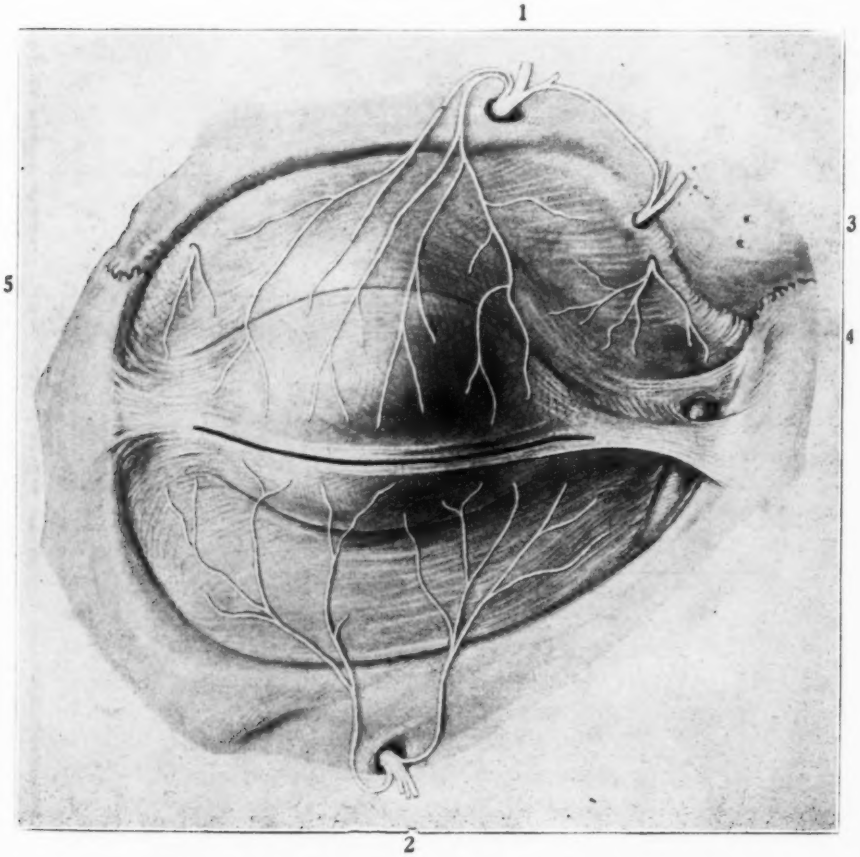

Figure 3. Anatomic distribution of nerves about the lids: 1, Supraorbital; 2, Infraorbital; 3, Supratrochlear; 4, Infratrochlear; 5, Lacrimal. All the main branches lie in the deeper layers next to the periosteum and the tarsoorbital fascia; hence the injection for blocking them should be in that deeper layer.

orbital nerve with an injection of the tissues between the nasal duct and the infraorbital foramen, so as to cut off the conduction of sensation in nerves which go from the neighborhood of the sac to the infraorbital nerve. Part of this injection should be subcutaneous, but most of it submuscular next to the periosteum, because the larger nerve branches run in that deeper layer between the orbicularis and periosteum. Next block off the supra- and infratrochlear nerves by a similar injection above the lacrimal sac. These three areas will give sufficient anesthesia for the dissection as far as the

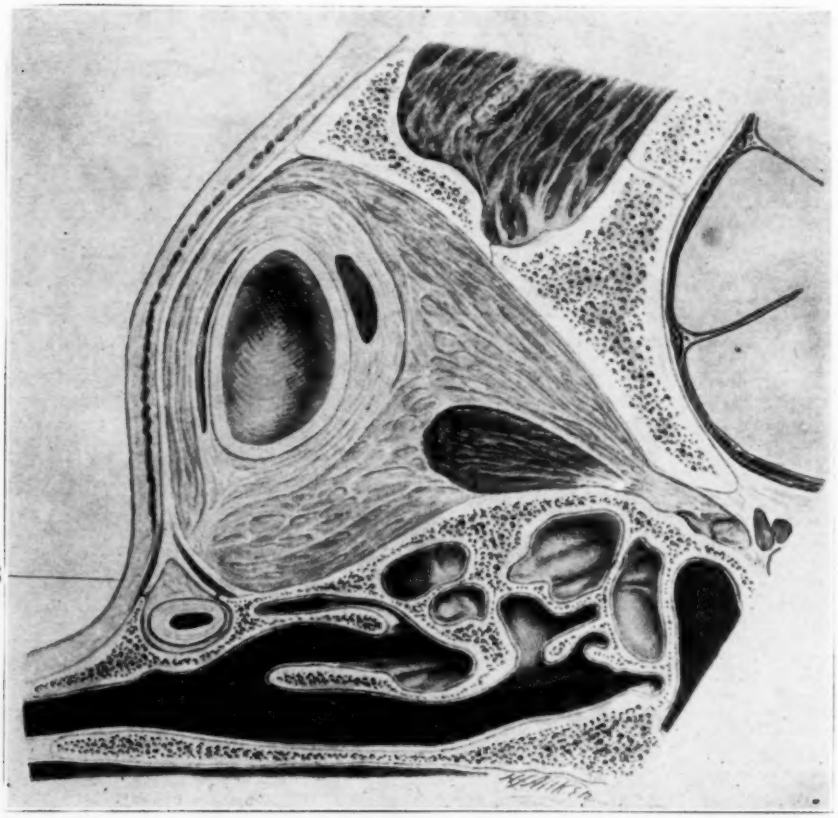

Figure 4. Cross section through the lower part of the orbit. S—Space around the lacrimal sac which should be infiltrated with novocain. Note how the tarsoorbital fascia divides into an anterior and a posterior layer, attached to the bone in front of and behind the sac. The bony attachment of the anterior layer can always be felt at the lower inner angle of the orbit.

deep fascia covering the sac. It it necessary to inject under this fascia and so into the areolar space around the sac before cutting the fascia, because this helps to keep the solution in the loose areolar tissue around the sac where it is wanted. Sometimes the needle can be inserted in the proper place before the skin incision is made. If not, it is usually pretty easy, after the skin and muscle are retracted and the bleeding stopped, to find the edge of the lacrimal groove in which the sac lies. At this edge, the deep fascia which covers the sac meets and coalesces with the periosteum which forms the inner wall of the space occupied by the sac. If the needle is insinuated through the fascia very close to the bone, it is usually possible to inject the solution into the loose tissue, where it spreads in all directions around the sac. When there has been inflammation and cicatrization, there may be no loose tissue. In this case, it is harder to inject around the sac. If the needle enters the sac, the fluid flows with little or no resistance, thus betraying its false position; also, the solution appears in the conjunctival sac by way of the canaliculi. If the needle is thrust too deep, entering the orbit, this is made known by the freedom with which the solution is injected; and the needle is withdrawn and inserted elsewhere, until the entire surroundings of the sac have been infiltrated. Lastly, the nasal duct should be injected. If it is found when the time comes to curette the nasal duct, that the anesthesia is incomplete at this point, a cotton wrapped probe moistened with 5 to 10% cocain and adrenalin may be held firmly pressed into the duct for a couple of minutes.

For operations involving the lids and adjoining tissues, for example, ptosis, entropion, ectropion, restoration of conjunctival sac with skin grafts, removal of growths and the like, one should follow the method of nerve blocking and supplement it by infiltrating the area of the operation. The nerves to be attended to are (1) the supraorbital, easily located at the supraorbital notch or foramen: (2) the infraorbital, which emerges from its foramen just below the lower margin of the orbit and on a line from the supraorbital to the 2nd bicuspid tooth; this too is easily located: (3) the supratrochlear and infratrochlear (see fig. 3). They emerge from the orbit at the upper inner angle, between the orbicularis and the bone, about halfway between the supraorbital foramen and the internal lateral ligament, just above and below the pulley of the

superior oblique. They supply the conjunctiva, caruncle, lacrimal sac, skin of lids and brow, and root of nose: (4) the lacrimal nerve in the upper outer part of the orbit supplies tissues in that region, especially the lacrimal gland and eyelid. The skin around the outer canthus and adjoining parts of the cheek is further supplied by the temporomalar branches, which perforate the malar bone from the orbit to the cheek.

These various branches of the fifth nerve supply the area in which most of our operating is done. It is possible to block the supraorbital and infraorbital at their foramina. It is not necessary to block the small and variable subbranches by so inserting the hypodermic needle as to actually hit their sheaths. The larger branches lie next the periosteum, and the tissue is generally a loose areolar tissue in which an injection spreads readily; so that if the needle is put fairly near the location of any of the larger branches, the solution can be made to bathe all the nerve trunks in that area. After the anesthesia has had time to penetrate the sheath and unite with the nerve in a sort of chemical reaction, the conductivity is effectually stopped.

A cordon may be drawn around any area which it is wished to anesthetize by injecting the solution in the form of a moderately broad zone in that layer in which the nerve branches lie. The nerves have to pass through this cordon to reach the area which we wish to anesthetize. In passing through the infiltrated zone, the nerves absorb enough of the procain to lose their conducting power, but it is necessary that the nerves be actually bathed in the solution.

To anesthetize the upper lid, for example for a ptosis operation, the needle should be inserted to the periosteum just below the supraorbital foramen, and one c. c. injected so as to spread for ten mm. each way. Another injection is made close to the periosteum of the margin of the orbit, near the pulley, and another below the lacrimal gland, blocking the outer end of the upper lid.

In the lid, itself, the larger nerve branches lie between the orbicularis and tarsus, sending twigs in all directions, especially forward to the skin. If it is desired to operate on a chalazion, the solution should be injected so as to surround the tumor on all sides, not so as to infiltrate the tumor itself. Then time should be allowed for the solution to penetrate the nerve sheaths so as to block them.

To secure skin for a skin graft, a large amount of solution is needed and a larger syringe is useful. Select the area for the graft, and then surround it with a liberal zone of endermal infiltration. It is necessary to underlie the skin with solution, also, as twigs come forward through the subcutaneous fat. Anesthesia is very good for about half an hour. Sometimes the edges of the conjunctiva begin to get sensitive in less than half an hour. This is in part because of the cutting of the conjunctiva and opening of the areolar spaces where the anesthetic has been injected permits this to be washed out by the blood, favored also by the manipulations, and in part because the returning circulation in this part of the conjunctiva carries away the anesthetic and restores sensation. Hence the insertion of the final stitches may cause pain. If it is enough to give trouble, you can stop and apply cocain and adrenalin to the particular areas where it is needed, using a pledget of cotton moistened with the solution and held in contact with the spot for 2 or 3 minutes.

The cases that are unsuitable for local anesthesia are those who are not controllable mentally, even with fairly liberal sedative treatment.

Very young patients. No definite age limit can be given. I have tried the ingenious suggestion of letting the very young patient get pretty hungry by deferred feeding, and then giving the bottle, which will often so engross the baby's attention that he will permit examination or operation under cocain.

Insane and feeble minded and hysteric patients often require general anesthesia. Some surgeons like rectal administration of ether in oil very much. I have no personal experience with it.

When there is infected tissue, which makes injection dangerous, one should not use local infiltration but blocking is not contraindicated.

I have not used local anesthesia for operations on bone.

It might be dangerous in certain very vascular tissue, which could not be blocked off but would require infiltration, the danger being of injecting into a blood vessel and producing toxic symptoms.

Finally, there are a few patients who have an idiosyncrasy toward cocain or procain, and are badly affected by it.

DISCUSSION.

DR. JOHN M. WHEELER, New York: One of the classifications of infiltration anesthesia is nerve block, which may be accomplished either by the introduction of the anesthetic into the nerve sheath itself, or about the nerve. The peridural method is the one usually advocated and the one of which Dr. Lancaster has spoken.

I did not hear Dr. Lancaster speak of any of the possible misfortunes which might come from regional anesthesia. I think it is important to speak of at least two. One is novocain, which is the usual anesthetic used for regional anesthesia. From experience, I know that there is danger of the surgeon getting into trouble by using a stale solution of novocain. The solution must be fresh.

Another thing which I think is quite important and which was not spoken of, is that there is genuine danger of injecting novocain solution into a vessel. There is always a possibility, in introducing the needle in regional anesthesia, of having the point enter a vessel, more particularly a vein, and if novocain solution in such amounts as Dr. Lancaster has spoken of is introduced into a vein and goes directly into the circulation, death may result—the heart may stop beating.

It has fallen to my lot to operate in a good many different hospitals, both general and special. I am sorry to say that it is a very common thing for the surgeon to be handed a syringe for use that is not in proper condition—extremely common. The point of the needle is likely to be dull, the connection between the needle and the barrel is likely to be bad, and the union between the plunger and barrel to be inexact, so that leakage and difficulty of manipulation are rather the rule than the exception. I think the surgeon should insist on the hospital furnishing him with the proper syringe and proper needle. There are several outfits that are all right, and I think you would be interested to know that Labat has just gotten up a set which is designed for eye, ear, nose and throat work. I have taken the liberty of having some slides made to show this outfit, so that you will have in mind at least that there is a dependable outfit on the market now.

The barrel, plunger, needle and nozzle are all mounted on a tray, so the whole outfit can be sterilized at once. There is a handle attached to the tray, so that it can be lifted out of the sterilizer readily, and this can be done even without sterile hands. The distinctive feature about this outfit is that the joint between the barrel and the cover of the barrel is easy of action, and yet is almost waterproof under pressure. The plunger is of metal and the barrel largely of glass. There is one caution, and that is that the barrel and the plunger should be of equal temperature, room temperature preferably.

DR. ELMER P. WEIH, Clinton, Iowa: Dr. Lancaster has given us a paper which has brought the subject up to date. It is a rather large subject, and he did not touch on all phases of the question. A number of things can be brought out: for instance, in doing an enucleation, if we will use a needle that is not too thick, the outer two-thirds of the needle being bent to approximately the same curvature as the eyeball; then by injecting just below the attachment of the external rectus, we can insert the needle very easily into Tenon's capsule and flood it. The fluid will

infiltrate entirely around the eyeball, thus making your enucleation absolutely painless.

Another point, if you wish to use a straight needle and make the injection deep, by following the external rectus straight back and also using a needle that is not too large, the contents of the orbit can be absolutely anesthetized by a surprisingly small amount of solution, if the solution is injected into the region of the ganglion. This method of injecting the ganglion is excellent in cataract work and in opening the posterior part of the sclera when one wants to use a magnet in the posterior part of the eyeball.

Dr. V. A. Chapman, Milwaukee, Wis.: I still object to the unfortunate word "procain." In the spoken word it is so much like "cocain," that there is likely to be dangerous mistakes made. I have drawn the attention of the Academy to this previously. In the written word, it is all right; but when used in the operating room, there is apt to be mistakes made. If we object to the word "novocain," it seems to me we should try to find some word that would be more easily distinguished by the ear in differentiation from the sound of the word "cocain."

Dr. Harry S. Gradle, Chicago: I do not think Dr. Lancaster spoke sufficiently at length of an important phase of anesthesia that may be used in removal of the eyeball. I refer to the true ciliary ganglion anesthesia used by Elschnig some years ago. In this anesthesia, the entire orbit and contents are anesthetized with a single injection, but the anesthesia may be used only when the eyeball is to be removed and when there is no further hope of vision. The usual local anesthetizing of the conjunctiva is carried out, and then the needle is introduced into the outer canthus below the superior rectus and directed backward and inward. There must be a mark on the needle indicating exactly 2½ centimeters. When this mark reaches the conjunctiva of the outer canthus, the tip of the needle should be against the inner orbital wall, perfectly horizontal, and the tip of the needle is then immediately in the region of the ciliary ganglion. Inasmuch as the ganglion is only one millimeter in size, one cannot hope to strike the ganglion itself, but by the use of between one and one and a half c. c. of 1 per cent novocain solution, sufficient infiltration of the region around the ganglion can be secured to produce complete anesthesia except in the lower third of the palpebral conjunctiva, which has a different nerve supply and which for complete anesthesia must be injected separately. Between five and ten minutes must elapse after the ciliary ganglion is injected before the operation is done. The great mistake is made in hurrying; time must be allowed.

There are certain conditions in which it is not wise to use an anesthetic like that—patients of an extremely nervous type. I do not think we are justified in using local anesthesia for that type of case.

One other point in Dr. Lancaster's paper that to my mind is open for discussion—where he mentioned injection for akinesia as well as anesthesia along the superior rectus and along the internal rectus in the operation for opening the eyeball. The less we can interfere with the orbital contents in cataract, in glaucoma operation and other operations for opening the eyeball, the sooner can that eyeball be set into position after the operation.

Dr. W. H. Snyder, Toledo, Ohio: As many of you no doubt will use this first for cataract extraction, I feel that a few practical notes might

be of assistance to you. After the patient is prepared—I speak of paralyzing the orbicularis—have the nurse make an application of iodin just external to the external canthus. You can then introduce the needle—I use a 22 or 23, 1¼ to 1½-inch long, with a point similar to that used in blood transfusion, a shorter point which will just engage the periosteum and not penetrate the veins. I sterilize my hands and introduce the needle, making three injections—one toward the ear, then turn the needle around without withdrawing and block the lower lobe, then upward to block the upper lobe; then withdraw the needle one-half and inject the balance. I use procain with adrenalin 1 to 1000.

Another very important feature is that you must wait ten or fifteen minutes before you begin the operation. You will have no edema, you will have a patient who will be just able to close the lid, but with no force. I have a finger on the point of the needle under the skin, and I have found it an advantage to simply put the needle under the periosteum and withdraw it a small distance, as I believe you get edema under pressure.

A very important point, which I have found from experience, is that whenever you paralyze the orbicularis, you must splint the lid or the patient will get the lid open under the bandage. I have had this happen twice. Fortunately, I had sense enough to take the bandage off. The patient said there was a feeling as if the eye was open. Usually one disregards this, but I opened the bandage.

I use adhesive strips, two or three on the upper lid above the lashes, and one or two along the lower lid below the lashes. Do not get them too wet. Then I put one or two crosswise over these to keep the lid borders approximated. That gives fair security that the patient will not get the eye open. There is a tendency for the eye to open, especially if you give a little too much anesthetic.

Dr. Walter B. Lancaster, of Boston, (closing): I want to thank the gentlemen who discussed the paper, especially Dr. Wheeler, for pointing out some very important things that I omitted. Some of the other omissions will be found in the paper itself.

Solutions should not only be fresh, and sterile, but free from sediment. A common fault of hospital solutions is the fine, floating particles. I reject any solution with particles in it. I think the formation of benign tumors is most likely due to sediment, and particles will cause that.

I did not emphasize the danger of rapid absorption, because I think we are all afraid of it, and I thought the most important thing to emphasize was the freedom from danger. If you have to use regional anesthesia by infiltration around a vascular tumor, where you are afraid there are large vessels that you would not encounter in the normal orbit, it is a good plan to do a little suction before you inject. That is, have your syringe not fully loaded so there will be suction when the piston is withdrawn. If you thrust the needle into a place where you fear you may have a vessel, pull back the plunger, and if there is blood, you are in a vessel. Then you must change the position of the point so as to be outside of the blood vessel.

The danger from cocain and procain is very greatly increased by rapid absorption, but the danger lasts only a short time. If you have a toxic case and can keep the patient alive less than an hour, the danger

is probably over. Quick absorption is of course retarded by adrenalin. Contrary to the advice of some of the most recent writers, I still advocate the use of adrenalin, at any rate in the orbit where the area is so small and hence there is no danger of a toxic dose of adrenalin.

The matter of the time to wait was gone into pretty fully by the committee of the American Medical Association, who suggested such a length of time as a half hour. In cataract extraction my custom is, after the subconjunctival injection, to wash out any novocain that may be in the sac, then fill the sac with mercurochrome and let that act while we are waiting; then after five or ten minutes, irrigate the sac, if necessary instil another drop of cocain for the local effect, because this is the important thing—as the operation goes on the anesthetic is carried away, and towards the end of the operation you have less anesthesia. If you have a flap in a plastic operation you may find the insertion of the stitches toward the end is painful, because the anesthetic has been carried away, so you have to supplement it with more cocain or novocain. I think, in most operations, ten to fifteen minutes is ample to wait, especially as the preliminaries to the operation take a few minutes, so by the time you get down to the actual operation, you have waited perhaps five or ten more minutes.

TWILIGHT SLEEP IN OPHTHALMIC SURGERY.

MEYER WIENER, M.D.

ST. LOUIS, MO.

Twenty-five years ago, Schneiderlin recommended the use of morphia and scopolamin in separate doses, as an anesthetic for operations. He administered to the patient, subcutaneously, 1½ to 2 hours before operation, scopolamin-morphia separately, which induced deep anesthesia. His work was followed up and elaborated upon by Korff. Schneiderlin recommended as high as 1/25 grain scopolamin and 1⅙ morphia in a space of 1¼ hours.

It was immediately taken up by various surgeons and both praised and criticized, according to what the individual results happened to be.

Von Steinbuchel supplied most favorable results on anesthesia obtained in this manner, but Schickelberger maintained that the morphia-scopolamin method was adapted only for those cases which rendered a general anesthesia absolutely requisite, and where at the same time chloroform or ether was contraindicated. The Schneiderlin-Korff method of anesthesia by morphia-scopolamin is based on the fact that the hypnotic and anesthetic properties of the two drugs associate themselves, while their toxic effects on the respiration and circulation, being antagonistic, counterbalance each other.

Stolz ascribed the tranquil nature of the anesthesia to morphia rather than scopolamin, and considered it was procurable, and with less danger, by injection of morphia or morphia with atropin, before administering an ordinary anesthetic. Wild and Flatau regarded it as risky and cautioned against its application.

Kionka attributed failures to impure preparations, faulty method, or applying the method to patients who are too feeble.

In Nicholson's opinion, no fatal case has been described in the literature which could be definitely proved to be due to the use of scopolamin. The lack of uniformity of action has been

ascribed by Ludenstein, in his report of 150 surgical cases, to individual differences.

Hatcher's report to the council of Pharmacy and Chemistry of the American Medical Association, in 1910, may be regarded as a summary of the situation up to that time. Scopolamin and hyoscyamin are considered identical. Statistics give 30 deaths in an unknown number of administrations. Scopolamin and morphia may cause death from paralysis of respiration. When this occurs, artificial respiration is usually ineffective and sometimes useless. Hatcher states that up to 1910, no death had been recorded in which the dose did not exceed 1/130 scopolamin and ⅙ morphia, although Ely reported the death of a patient from 1/100 and 1/8. Contraindications are when respiratory function is decreased, all cardiac diseases and other correlatives which interfere with the circulation, also Graves disease. His conclusions are that:

(1) An attempt to use scopolamin and morphia alone, without the addition of some other drug, is dangerous.

(2) There is no possible excuse for ready made mixtures of scopolamin or morphia. They should be prescribed separately, according to the patient's condition.

Krebs and Wilson reported the second thousand deliveries in Barnes Hospital under scopolamin and morphia seminarcosis. The dose was 1/133 scopolamin and 1/6 morphia. A second injection of scopolamin is given in 45 minutes. If necessary, a third injection of scopolamin is given in 45 minutes. They test the degree to which the patients are under by a very simple but accurate method. Before the second injection, and before each subsequent contemplated injection, the patient is requested to put her index finger to the tip of her nose, the eyes being covered. If she succeeds in doing this promptly, she still retains locomotive coordination, and the contemplated injection is given. If, however, she moves her finger around vaguely and misses the mark, she has lost locomotive coordination and the injection is omitted, or the dose reduced, for the patient has the desired amount of the drug. In most cases, this stage is reached shortly after the third injection, but not in a small number shortly before the third. After the third injection, most patients remain sufficiently scopolaminized for two hours.

Strobell used scopolamin and morphia in the field, during the war, for emergency operation, making the patients more

comfortable for transportation, producing no nausea or vomiting, and requiring no nurse at the bedside, the patient awakening rested and able to take nourishment. He gave 1/100 scopolamin and ¼ morphia, repeated in 30 minutes, and in ½ hour again gave a 1/200 scopolamin and ⅛ morphia.

Hurd reported morphia-scopolamin in a few cases of nasal operations, and later as a preliminary to local anesthesia, relating eight years' experience. Before its use, he had had several cases of poisoning from cocain and the local anesthetics. With the morphia-scopolamin, he can use more local anesthetic. It eliminates apprehension on the part of the patient and blocks the memory, but the patient is not always ready for the operation at the appointed time. He used morphia ½ and hyoscyamin 1/100. He had two cases operated in sitting posture, where the blood pressure fell to 90 mm. He claims good and bad risks react alike. His paper was discussed by five others, mostly favorable and agreeing in general.

In the report here submitted, 93 patients were given morphia-scopolamin in conjunction with local anesthesia. Most of these were strabismus cases, where advancement of an ocular muscle was made either with or without lengthening of the opposing muscle. There are 61 muscle cases; 20 glaucoma; 3 entropion, and 6 dacryocystitis, and one case each of conical cornea, enucleation and xanthoma. As a whole, the results have been most satisfactory, with no known serious complication, unless it be one in a case of glaucoma which will be mentioned. In muscle cases, it is most desirable to have the cooperation of the patient as well as anesthesia, which one cannot obtain with a general anesthetic, and seldom with a local one.

All of the operations were performed at the Barnes, Jewish, Children's and Bethesda Hospitals.

Most of the 61 muscle cases were in children, who would otherwise have been subjected to the risks of a general anesthetic. Some of them probably could have been more easily performed than with unsuccessful twilight. I think that our great fault in the beginning was in not waiting long enough for the morphia-scopolamin to take effect. We found our best results were obtained when we waited between 2½ to 3 hours after the first dose. Also, other things being equal, the younger the individual the less quieting the effect of the morphia-scopolamin. The most effective final adult dosage

was found to be morphia ¼ and scopolamin 1/130, followed in 45 minutes by 1/200 scopolamin and 45 minutes later by 1/200 scopolamin; the operation starting 2½ hours after the first dose of morphia-scopolamin. Only one dose of morphia is given. The dosage for children was given in proportion to their age.

In 58 cases, or 62%, fairly complete analgesia and amnesia were obtained. Good amnesia with poor analgesia was obtained in 8 cases, or about 9%, and about the same percentage with poor amnesia with good analgesia, as well as both analgesia and amnesia fair. Three cases result in complete failure, ether being administered to complete the operation.

The most successful cases were the patients operated for glaucoma, the reason being, most likely, their advanced years.

One case of glaucoma developed a choroidal hemorrhage, with the loss of vision three hours after operation. This undoubtedly could have occurred with only local anesthesia, but as the patient was very much excited from the scopolamin, it probably was a contributing factor. This is the only patient where the morphia-scopolamin was used, in which I could blame the twilight for a bad result. Absence of pain, or at least cooperation of the patient with little or no manifestation of pain, was especially observed in the six cases of dacryocystorhinostomy, where it is necessary to have the patient in the sitting posture and the local anesthesia alone is not entirely satisfactory.

The plastic operation on the cornea is better without the morphia-scopolamin, as there is ordinarily little or no pain with local anesthesia, and one does not care to risk the lack of a complete coordination of the patient which is so essential.

The entropion cases were entirely successful, as was the one case of enucleation.

Various doses and combinations of both morphin and scopolamin were tried, but the most successful, and that used at present, is morphia grain ¼, scopolamin 1/130 (B. W. ampule), followed by 1/200 scopolamin in 45 minutes, and another 1/200 in 45 minutes, the operation starting about 2½ hours to 3 hours after the first injection. Care must be taken to avoid noise, light and outside influences after the twilight is started. In advanced adults, the patient often is brought into the operating room in a deep sleep, from which it is difficult to arouse him.

CONCLUSIONS: Twilight sleep is successful in producing satisfactory amnesia and analgesia, in conjunction with local anesthesia, in over 60% of cases tried, and in at least 80% of the cases is more satisfactory than local anesthesia alone, in cases of strabismus, glaucoma, dacryocystitis and entropion.

There is probably more danger of expulsive hemorrhage in glaucoma than without, unless the dosage is most carefully watched. In the future, I would refrain from operating on a patient with glaucoma who came to the operating room in a stage of excitement, and would rather postpone the operation. The dilatation of the pupil resulting from the scopolamin is no disadvantage, as it has always been our custom to instill atropin in these cases. Most of the patients have been pleased with their experience, and would not hesitate undergoing the operation under similar conditions.

The great advantage in cases of strabismus over a general anesthetic, where cooperation of the patient is such an advantage, is self evident. While I am sure we still have much to learn in its administration, I feel that it should be given a thorough tryout.

DISCUSSION.

DR. WILLIAM L. BENEDICT, Rochester, Minnesota: There is no doubt that patients going into an operating room with a composed attitude and with confidence that things are not going to be so bad—that they are not going to be hurt—is a very desirable thing to bring about. Any method that will inspire confidence in a patient when he comes into the operating room is worthy of trial in those cases in which we believe it will work successfully. I believe, first of all, that we should attempt to inspire confidence in the patient by conversation, by attempting to acquaint him with things about the hospital, attention by the nurses, lack of distracting noises, and the avoidance of any procedure that is likely to excite suspicion, such as the administration of hypodermics, or the useless giving of baths and enemas, alcohol rubs, useless taking of temperature, and things of that kind. Most of our people are unacquainted with hospital usages or hospital procedure, and if too much formality is shown, too many things done to the patients which they do not understand, it only increases their apprehension and militates against their coming into the hospital in a frame of mind conducive to calmness. I do not think it necessary to go to the extreme of operating patients in their own bed, because I think most of us will agree that surgery should be done in an operating room, where conditions of surgical procedure can be standardized and made more favorable. In a selected number of cases, the administration of drugs to bring about composure is admirable and in some cases advisable. But Dr. Wiener has pointed out that in some cases it is not advisable to use scopolamin or morphin.

Scopolamin and morphin have been well discussed before this Academy and other societies. It is well known that the administration of from 1/150 to 1/300 grain scopolamin will produce in most people, particularly those who are more or less apprehensive, a relaxation that will last sometimes over a period of 25 to 45 minutes. In observing a large number of patients in psychopathic hospitals a few years ago, I found that the height of excitability following the use of scopolamin was 25 minutes. Dr. Wiener has advised giving scopolamin 2½ hours and 45 minutes before going into the operating room. With most people, the height of excitability will have passed by this time, and they should be in good condition for the best results from using the drug.

I think it is inadvisable to use this narcosis on individuals who are to have cataract operation, or those in which we must depend upon voluntary action during the operation. His test of fingers and nose applies very well for the coordination of the eyes in holding the position required by the surgeon. The voluntary movement has not disappeared, and if given time they will perform the finger-nose test very satisfactorily. They will also put the eye in proper position, but their conception of the duration of time and also of the coordination time, that is to say, as to when to perform a given movement after having been advised to do so, seems to have left them, and when told to look up they may do so quickly, or they may be several seconds, or until the request has been repeated two or three times. Then instead of holding the eye in position they are likely to move to one side or the other.

Furthermore, those individuals who are to have general anesthesia are probably more in need of a sedative than those who are to have local anesthesia. The use of gas for general anesthesia has largely done away with the period of excitability produced by the use of chloroform or ether, and with the new methods of giving carbon dioxid and ethylene with an experienced anesthetist, there may be no period of excitability either before or after the anesthetic.

For one year, we used routine administration of morphin and scopolamin in our eye clinic. That was three years ago. All cases in which it was not contraindicated were given morphin and scopolamin, not exactly as Dr. Wiener has stated, but before operation. We found the period of excitability before operation, the lack of coordination in the operating room, and the inability of patients to hold themselves properly at rest in bed afterwards, was more than without it, and we have discontinued it almost entirely. A short time ago, we used scopolamin and morphin on a man of about 60, who had been under treatment for corneal ulcer for several weeks, and who had a cataract in the other eye. He had been very tractable and there was no concern whatever. Within two hours after his operation, he became so disorientated that it was necessary to keep two nurses on duty to keep him quiet enough for the safety of his convalesence.

DR. W. H. WILDER, Chicago: I cannot speak with any authority on this subject, because I must confess that I have been a little hesitant about using it. I recall too vividly experiences we had some twenty-five or thirty years ago with scopolamin as an aid in refraction. Some of these experiences were so unpleasant, including such incidents, for example, as a patient attempting to jump out of a street car window after leaving the

doctor's office, another of wild delirium, that I have been reluctant about using it to produce twilight sleep, fearing that possibly the twilight might merge rapidly into night. But I do not wish to speak in any critical way of this paper, which is excellent, and think it is proper enough to try out this method. I think we are all agreed that if we can get a happy state of confidence and feeling of security and comfort of the patient, we have accomplished a great deal in the way of preparation for the operation.

Alluding to what Dr. Benedict has said, I think we are often too fussy in our operations; with too much preliminary work and too much preparation and palaver in the presence of the patient, which tend to make him nervous and apprenhensive.

I am satisfied from personal experience, that it is excellent practice to get our patients into a comfortable state of mind, and in many cases we can accomplish much by having them in the hospital the day before the operation, getting them acquainted with their surroundings, giving a good night's rest with reliable hypnotic, such as chloral and bromides. The method that Dr, Lancaster speaks of as mental anesthesia I think is excellent.

I would hesitate to use morphin and scopolamin in combination in case of cataract, because my experience in the use of morphin before cataract operation has been so unpleasant. You never know when it may produce nausea and subsequent vomiting, which of course is undesirable. But it seems to me it is desirable to induce, not necessarily a complete anesthesia, as in twilight sleep, but a partial degree of hypnosis, so that the patient will lie quietly on the table and yield more readily to the local anesthetic we use, and this may be accompished with some reliable hypnotic, other than morphin, administered a couple of hours before the operation.

I am a warm advocate of the value of infiltration anesthesia, particularly blocking of the orbicularis muscle, which to my mind is the one that more frequently jeopardizes the cataract operation by its violent contraction. I usually follow this with infiltration around the ciliary ganglion, possibly 1 c. c. of 2 percent. solution of novocain. In the injection into the orbit, I try to avoid infiltrating any of the muscles of the muscle cone, because it seems to me, in case of cataract extraction, that we do not want to block these muscles, at least not completely, as we desire the cooperation of the patient in moving the eye.

In this I agree with Dr. Benedict, that we need the cooperation of the patient, not only in cataract, but in the strabismus operation.

Dr. Lee M. Francis, Buffalo: Until seven years ago, I had had some very unfavorable experiences with nausea following the use of morphin. Under the advice of one of our good clinicians in Buffalo, I have substituted the following: Three hours before cataract extraction, the administration per rectum of 20 grains chloral hydrat combined with one dram of sodium bromid. In these years, I have not had postoperative vomiting that could be attributed to the anesthetic or preparation.

Dr. M. W. Jacobs, St. Louis: I think I have had opportunity of using twilight sleep in a sufficient number of cases to have come to the conclusion, that the difficulty in the past has been the matter of dosage, and while the essayist will probably bring out this matter in the closing of the discussion, I want to voice the opinion, that with the dosage as

worked out in the more recent cases, the percentage of successes would have been greater than 60 per cent, as shown by Dr. Wiener's paper. I believe the last word has not been said on the use of twilight sleep, particularly in strabismus cases in young children. While I have had a few rather disagreeable experiences with twilight sleep, I have had a sufficient number of successful anesthesias by using this type of narcosis to believe that if the dosage is eventually worked out, as I hope it will be, we will have a narcosis which is of real value, and which should not be shelved without further investigation and trial.

Dr. Edmond E. Blaauw, Buffalo: This subject is a great one, and I would like to endorse all that has been said, especially by Dr. Wilder. I like to give O. S. adalin the evening before the operation, and another similar dose two hours before the operation. Those of us who have not large surgical material should try to remain on the safe side.

I would like to refer to the rectal hedonal administration used in the clinic of Axenfeld. You will find it in the Klin. Mon. of Aug. 1924, Bd. 72, p. 195, (G. Sondermann). I think it is a valuable suggestion when children should be kept quiet for a long time repeatedly, as for X-ray treatment.

Dr. Harry A. Smith, Delta, Colorado: For over five years, this mode of anesthesia has been the routine in our work. For a long time, the weight and age of the patient was recorded and the dosage worked out on physiologic principles. The dose should be regulated on physiologic principles to get the result that Dr. Wiener has been talking about, but it is not a mathematical problem, it is art. If you want results in these cases there is just one way to get it, and you can do it in a much higher percentage of cases than Dr. Wiener has mentioned, namely, 65 per cent. But it takes close attention, by yourself or by a trained assistant who knows the condition of your patient from the time of the first dose, usually 30 to 60 minutes before operative procedure is to begin. This cannot be done by a rule of one-fourth grain or any other amount. It must be done by the observation of someone trained to do it.

The mastoid operation is the only one in which we do not use modified twilight sleep. We have used one-eighth grain dose in a woman 70 years old, removing an abscess of the lacrimal gland and anterior ethmoids, and when she awoke three hours after the operation, she did not know she had been operated on. We have given one and a half grains to a highly nervous patient. It is an art, and the one who watches the patient must be trained for that particular kind of work. I think in that way Dr. Wiener would soon have 98 per cent success in these cases.

Dr. Meyer Wiener, St. Louis, (closing): Dr. Benedict and Dr. Wilder spoke of the anxiety of the patient as something that should be allayed. That is one of the reasons that we use twilight sleep, but not the main reason. The main reason is that we want the cooperation of the patient; we want the patient quiet, and we feel that if we can get the cooperation of the patient and get him to coordinate we can direct his eye by suggestion. I know that if I tell a patient on whom I am performing a muscle operation under twilight to look up, he will look up, although he may take a little longer time than usual; but he will not continue to look up unless I continue to tell him to look up. One can quietly suggest to this patient that he continue to look up. You will have to repeat it

several times, because these patients are open to suggestion, and more so than patients under ordinary conditions. I have little difficulty in securing the cooperation and coordination of the patient in twilight, and that is the main reason I use twilight sleep. This I cannot get with general anesthesia.

Dr. Wilder's fear of twilight sleep fading into night I think is unfounded. I have not had any suggestion of danger to the general condition of the patient from twilight, and the experience at the Barnes Hospital in obstetric cases is, that in nearly 3,000 cases, there has not been one case in which death could be attributed to twilight. They give a very much larger dose than I do, but they watch their patients very carefully.

I did not report any cases of its use in cataract extraction because I do not think it is indicated in cataract. With the ordinary instillation anesthesia plus occasional infiltrations of the conjunctiva with novocain, we can get enough anesthesia to keep the patient quiet, and there is, as Dr. Wilder has said, a great deal in the proper handling of the patient before he goes to the hospital, and not too much done after he gets to the hospital. I agree with all of the men thoroughly on that.

I do not know anything about the rectal injections which Dr. Francis mentioned.

The dosage, as Dr. Jacobs stated, is very important. Dr. Smith feels we will get a larger percentage of successes. We have a larger percentage of successful twilights, but the 65 per cent includes cases that are successful in every way. We have had at least 80 per cent that were more satisfactory than the operations would have been without twilight.

As far as dosage is concerned, Dr. Smith stated that we could not give dosage by the rule of three. There is no mathematical way of regulating the dosage, because some people are more susceptible to morphin and hyoscin than others. But we can regulate it in a way, by the weight of the patient and also the age. The older the patient the smaller the dosage, and especially in very old people we give much smaller doses, and perhaps one small dose will more than suffice.

As to the patient being obstreperous after the operation is finished, if the patient is not unduly excited within two and a half or three hours after the twilight has been given, in my experience, he will not be excited after operation. I have made the mistake of operating on a patient when he came to the table excited. I shall not do it again. If the patient is not perfectly quiet when he comes to the operating room, I do not think he should be operated upon, but should be sent back, and the operation delayed.

CHOKED DISC AND VITREOUS OPACITIES FOLLOWING FRACTURE OF THE SKULL.

CLARENCE LOEB, A.M., M.D.

CHICAGO.

The presence of choked disc following fracture of the skull and intracranial hemorrhage is not uncommon. In fact, it is always looked for, and, when found, is of the greatest diagnostic importance. The appearance of retinal hemorrhage, with or without choked disc, in skull fracture is much rarer, and rarest of all is the presence of vitreous opacities.

In regard to the latter, the American Encyclopedia of Ophthalmology[1] says: "Interesting are those hemorrhages which occur after cutaneous burns, and those which follow compression of the thorax and of the neck, and blows upon the head. These hemorrhages may be in any layer of the retina, may remain in their original position, or may burst through the lining membrane and fill the vitreous." Fox[2] says: "Hemorrhage into the vitreous is always derived from the vessels of the retina, choroid or choriocapillaris. Injury to the head or eyeball is the most frequent cause of this condition. Oftentimes, the hemorrhage is so large that the chamber is filled and the retinal reflex is absent or obscured." It will be noted that no mention is made in either reference of the presence of choked disc.

In this connection, two interesting cases have been reported, where the element of trauma was lacking, but where vitreous opacities were present accompanying increased intracranial pressure. Dupuy-Dutemps[3] reports the case of a woman who had an apoplectic stroke, followed by coma. The first examination of the eyes, three days later, showed large vitreous opacities with reddish reflex on oblique illumination. Since the condition of the disc is not stated, it is possible that it could not be made out on account of the vitreous opacities.

Van der Kooi[4] reports a case where the patient had been haying all day, following which he became unconscious, with Cheyne-Stokes breathing and convulsions. Rochat examined

his eyes and found blood clots in the vitreous. So far as could be seen through them, the retina was normal. In this case, also, the condition of the disc is not stated, but since the retina could be seen at least dimly, it is reasonable to conclude that there was no choked disc.

As to the cases with choked discs and retinal hemorrhages, without vitreous opacities, a few references will be sufficient. Dupuy-Dutemps[3] quotes several cases where there were papilledema and retinal hemorrhage, with hemorrhage into the sheath of the optic nerve, following intracranial trauma or other causes.

Knies[5] says: "If an intracerebral hemorrhage occurs. . . . the blood may enter the sheath of the optic nerve and produce changes that are visible with the ophthalmoscope (choked disc) . . . Choked disc, with or without retinal hemorrhage, is occasionally observed."

Panas[6] reports four cases of cranial trauma followed by bilateral hematoma of the optic nerves and edema of the discs, with retinal hemorrhages, seen during life.

De Schweinitz and Holloway[7] report a case where the patient was found unconscious. Ophthalmoscopic examination showed hemorrhages in the retinas of both eyes. Postmortem examination showed fracture of the skull, intracranial hemorrhage, and the sheaths of both optic nerves distended with blood. They, also, review the literature.

From these and similar cases, it would seem that the choked disc which follows fracture of the skull is not due to increase of intracranial pressure, but is rather an actual penetration of the hemorrhage into the sheath of the optic nerve, where it causes a papilledema by interfering with the return circulation. The retinal hemorrhages are the result of rupture of the retinal veins, or a penetration of the hemorrhage from the sheath of the nerve into the retina.

In none of these cases, and in none of the case reports in the literature to which I have had access, has there been the syndrome of skull fracture, choked disc and vitreous opacities. The infrequency of vitreous opacities following cranial trauma is evidenced by the paucity of references in the literature, which of itself would make the reporting of an additional case worth while. Furthermore, on discussing my case with several other oculists, I could find no one who could recall a case where the combination of choked disc and vitreous

opacities followed skull fracture. This fact added to the further one, that I could find no case recorded in the literature, is the reason for reporting the present case.

Case Report.

Mr. M. L. N., white, age 32. In the latter part of 1923, the patient was thrown to the street by the sudden starting of a car which he was boarding. He was picked up unconscious, and taken to a hospital, where he remained in a stuporous condition for some time. His history during this period was not available, but so far as I have been able to learn, the treatment was expectant, and there was no examination of the eye-grounds. An X-ray picture, taken shortly after the accident, showed a fissured fracture of the left side of the head, radiating from the vault to the base, through the frontal and parietal regions.

About three weeks after the accident, Dr. Harry Jackson was called into the case. His examination showed extreme tenderness over the left side of the head. The pupils reacted to light and accommodation, but the vision was very poor in each eye. There were no focal paralyses. The cremasteric and abdominal reflexes were absent on both sides, with a bilateral Babinski and ankle klonus. The spinal fluid was slightly yellow in color, and under increased pressure (20 mm. Hg.) After several lumbar punctures, with withdrawal each time of 20 c. c. of spinal fluid, the pressure returned to normal.

Dr. Jackson's report of the present condition of the patient, dated 18 months after the accident, states that, "The patient fatigues quickly, and has some tremor of the right hand. Babinski is no longer positive. He is unable to stoop or lift heavy objects. There is a considerable tendency to fits of despondency, and he is easily excited and becomes angry on slight provocation. Noises irritate him."

I was called to see the patient Dec. 10, 1923. Examination showed both eyes normal externally, with pupils dilated from the use of homatropin. A fair fundal reflex, obscured by opacities in the vitreous, was obtained from the right eye. View of the fundus was blurred because of large and small opacities, but it could be seen that the disc was in the stage of receding papilledema. In the left eye, the fundal reflex was obtainable only when the eye was turned to the extreme left.

The vitreous was almost completely filled by a large mass of blood clot, possibly hiding a detached retina.

Vision could not be taken in either eye, but it was roughly large objects in the right and perception of light in the left The treatment consisted of hot compresses of boric acid solution and one drop of 5% dionin twice daily.

Two weeks later, the right fundus was still hazy on account of vitreous dust. In the left eye, there was a good reflex from the temporal side, but the nasal presented a grayish appearance similar to a detachment of the retina. The dionin was increased. Four weeks later, both eyes were improved. In the left, it was evident that there was no detachment of the retina, but the disc still could not be seen. Refraction gave

V. O. D. = 6/12 nearly; with + 1.25 cyl. ax. 105 = 6/8.

V. O. S. = Fingers 1 M.; with + 1.0 cyl. ax. 90 = 1/12.

These were prescribed, and the dionin increased to 10%.

March 7, 1924. V. O. D. = 6/8, no improvement with glasses. V. O. S. = 1/15, no improvement with glasses. Opacities decreasing in size and number. July 25. V. O. D. = 6/6 nearly, no improvement with glasses. V. O. S. = 6/60; with —0.5 = 6/20. Aug. 28. Patient complains that at times a veil comes in front of the right eye, which is probably due to the accommodation, as there are no gross opacities. There is a large one in the left eye, directly behind the pupil, which prevents a view of the disc. On directing the eye to any side, the fundal reflex can be obtained. Under homatropin— V. O. D. = 6/15; with + 0.75 S. ⌒ + 0.5 Cyl. ax. 90 = 6/6 with difficulty. V. O. S. = 6/60, no improvement with glasses. Fundus and vitreous of right eye normal. Fundus of left eye dimly seen through large and small opacities.

Since the above date, he has been seen several times, the condition of the right eye remaining normal and the opacities in the left gradually becoming less. However, owing to the central position of the large opacity, the vision in this eye is about 6/60. He is able to do his work, but complains at times of a veil in front of the right eye. The treatment has consisted of hot compresses, with dionin in increasing strength, up to 20%. At no time have I seen in the right eye any area indicative of recent or earlier retinal hemorrhage. The vitreous opacities in the right eye are certainly, and in the left eye are probably, from hemorrhage into the vitreous by way of the sheath of the optic nerve.

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DISCUSSION.

DR. HARRY JACKSON, Chicago: I wish to thank you for the invitation to address the members of the Academy of Ophthalmology and Oto-Laryngology, especially so, since the subject is one in which I am greatly interested. I have the opportunity each year of seeing several hundred cases of injuries to the head at the Cook County and Michael Reese Hospitals.

The complications of head injury are often referable to the intracranial hemorrhage and edema of the brain. The late findings often include hemorrhagic cysts or persistent edema of the brain, and these, in turn, cause many focal lesions, including loss of sense of smell and taste, paralyses of the extremities or motor speech aphasias. Mental symptoms are common, also.

In the past, I have always made it a rule to examine the eyegrounds for early evidences of choked disc, which is so commonly seen in severe injuries to the head. In over 1500 cases seen in the past eight years, where there has been blood in the spinal fluid, giving evidence of subdural hemorrhage, the case reported by Dr. Loeb is the only one where the eye was not directly injured that was associated with marked vitreous opacities. I, therefore, agree with him when he remarks that the association of skull fracture and brain injury with choked disc and vitreous opacity is most rare.

Of late years, I have made a practice of performing spinal puncture in cases of head injury, to determine the pressure of the cerebrospinal fluid and blood content, basing a diagnosis of brain injury on these findings rather than waiting for choked disc or other symptoms to appear, as these latter symptoms usually appear too late to be of value for treatment of edema of the brain.

The removal each day of 15 to 30 c.c. of bloody spinal fluid so relieves the subdural pressure in edema of the brain following the injury, that choked disc and other medullary symptoms usually do not occur. In the treatment of this particular patient, it is possible that had the case been seen early, the administration of magnesium sulphat or the performance of lumbar puncture with removal of blood and spinal fluid might have influenced favorably the degree of choked disc and the amount of the vitreous hemorrhage.

Dr. F. Phinizy Calhoun, Atlanta, Georgia: I think it was Leslie Paton who reported a case of transverse subarachnoid hemorrhage in which papilledema did not occur for several weeks. It was his belief that the hematoma had become encysted and acted as an intracranial tumor.

Whether the occurrence of papilledema in these cases is sudden or delayed, I believe the mechanical cause to be the same, that is, an overdistention of the nerve sheath by blood or arachnoid fluid, which interferes in the return venous flow of the retina.

The retinal hemorrhage, formed as the result of the rupture of the dilated veins accompanying the papilledema, seems to be analogous to the picture of thrombosis of the central retinal vein.

Of particular interest in these cases is the formation of retinal hemorrhage independent of papilledema. I have not shared the belief that they were due to the penetration of blood from the sheath of the nerve into the retina, which as I understand is Dr. Loeb's idea, and which is supported by Dr. Paton, who thinks that this blood may pass into the retina by way of the perivascular lymphatic spaces which surround the disc. The existence of such spaces large enough to contain blood has been denied by Riddock and Goulden, who had the opportunity of examining histologically such an eye under consideration. I believe the cause is the same as where papilledema is present, and the retinal hemorrhage alone merely represents an early stage, and that the absence of disc changes is evidence of rapid absorption of a not too large clot in the subarachnoid space of the nerve, and the restored venous flow to normal.

Of great interest, and of diagnostic importance in all cases of subarachnoid hemorrhage, whether traumatic or spontaneous, is the pressure of blood in the spinal fluid.

FIELD AND FUNDUS CHANGES OBSERVED IN CASES OF ENDOCRINE DYSFUNCTION.*

WILLIAM D. ROWLAND, M.D., F.A.C.S.

BOSTON, MASS.

In an impartial, routine ophthalmologic examination of a considerable series of cases, investigated for the possible presence of diseases of the ductless glands, it was noted that *enlargement of the blind spots,* and *yellow discoloration* of the *nerveheads* was frequently found, while, with less frqeuency, contraction or alterations of the form and color fields was observed.

The present, preliminary, paper embodies an analysis of the eye findings in two hundred eighty (280) cases where an endocrine diagnosis was established with certainty.

The cases here reported are drawn from a much larger number referred to the Evans Memorial for diagnostic study. Preliminary reports of the methods employed and the results attained have already appeared in the literature from Rowe[1-12] and his associates, and an extended report based upon the study of a series of one thousand (1,000) cases is now in preparation. Briefly, it may be stated, that a long series of socalled vital function tests have been standardized by application to groups of demonstrably normal men and women. These tests, supplemented by very complete clinical observations, were next applied to individuals of established endocrine etiology, such as surgical castrates, hypothyroid and pituitary subjects, and advanced cases of exophthalmic goiter and acromegaly. The direction and amount of deviation from the normal induced by the several conditions in the complete series of observations, gave the basis for the differential diagnosis of the individual endocrine entities. The third group, from which this series is drawn, comprised, in the main, cases of an obscure etiology which presented various clinical evidences of a possible endocrine involvement. The clinical study and laboratory observations were sufficiently compre-

*From the Evans Memorial, Boston, Mass., No. 105-A-53.

hensive to evaluate existant nonendocrine pathology, and only in the proven absence of any such conditions were observations interpreted in terms of endocrine disorder, the elimination of all cases but those not explained by a known cause, other than the endocrine disturbance, should be emphasized.

The value and significance of ophthalmologic examinations was recognized from the beginning, but for a variety of reasons they could not be applied in all cases of the first series. The present group of two hundred eighty (280) cases are drawn from the first five hundred (500) in whom an endocrine diagnosis was established. Subsequently, as conditions have permitted, the eye examination has been made an integral part of the routine examination of every case. The earlier examinations brought out some highly interesting facts. It was early noted that many cases exhibiting enlarged blind spots and yellow discs, proved in the ultimate analysis to be classified as some one of the several forms of pituitary aberrations. Field changes and other disc changes have been frequently encountered, as well as vascular and pigmentary alterations.

An important fact to carry in mind is, that these cases have been examined impartially, no previous knowledge of the patient having been obtained; therefore, the findings are purely physical findings per se. Subsequently, certain history and subjective symptoms were ascertained to aid in interpreting questionable points.

The cases were studied under two groups: the B series, which entails a study for a period of one week, and from which one hundred fifty-five (155) of the cases here presented are drawn, and the S series, composed of cases receiving an abridged form requiring but half of one day, which furnishes the remaining one hundred twenty-five (125). The routine ophthalmologic examination applied to these cases included the visual acuity, the size, shape, and reactions of the pupils, extraocular position and motions, a study of the media and fundi, and field study including form, colors, blind spots, and maculae.

TECHNIC. Peter's hand campimeter was used for blind spot, form and color field determination. Using Peter's blind spot delineation, any measurement in excess of a circumferential two degree, or a quarter, half, or three-quarter three or four degree enlargement are classified as "enlarged blind spots." All enlarged blind spots thus obtained were further

checked on a small Lloyd slate with a wide angled stereoscope. Macular acuity studies were made with the latter instrument, using a 1.5 mm. object at 16.5 cm. for white, blue, red, green. The test object was carried from blind to seeing, and signalled usually by voice. A 5 mm. or 6 mm. object was used for form and color fields, and a 3 mm. object for the blind spot determination. With the hand campimeter of Peter, the latter sized object subtends an angle of two degrees. Glasses were not used except in rare instances of high refractive error. The hand campimeter was used because a portion of the patients were seen at the bedside, several in fact in bed, and not infrequently partly reclining or prone, while the majority were studied in better office circumstances; a uniform laboratory technic was thus rendered impossible. Attempts were always made to obtain uniform lighting, so far as possible, and the type and degree of illumination was always noted on the record. Ophthalmoscopic studies were made with an unscreened battery ophthalmoscope, freshly activated, thus insuring uniform light intensity.

The results thus obtained can be presented most succinctly in tabulated form, classifying on the basis of the endocrine focus involved.

TABLE 1. THYROID DYSFUNCTION. SIXTEEN (16) CASES IN 155 B SERIES.

Positive eye findings shown in 7 cases:

Enlarged Blind Spots	5 cases
Contracted Fields	4 cases
Vascular Changes	1 case
Yellow Discs	1 case

Percentage of positive eye findings = 43.7.

Of the five cases showing enlarged blind spots, three were bitemporal enlargements with normal nasal borders. The enlargements were all moderate, two degrees to five degrees at the widest point. Color fields were contracted concentrically in one case, while form fields were cut above in three cases. All seven cases showing positive eye findings were hypothyroid in type, and heavy lids obstructed upper fields somewhat. One case is of special interest, No. 110, (Fig. 2), which exhibited upper field cut to twenty-five degrees, and

which recovered full fields in three months under thyroid therapy.

TABLE 2. GONAD DYSFUNCTION. 28 CASES IN 155 B SERIES.

Positive eye findings shown in 10 cases:

Enlarged Blind Spots	10 cases
Yellow Discs	3 cases
Contracted Fields	5 cases
Pigmentary Changes	1 case

Percentage of positive eye findings = 35.7.

Nine of the ten cases exhibiting positive eye findings were females of hypogonad type. Blind spots were all moderate enlargements except one case, which had a right blind spot enlarged to eleven degrees diameter, and a left enlarged to nineteen degrees diameter. Field changes were all concentric color contractions, except the one showing the very large blind spots, who had also some form field contraction. The pigmentary change was a "snuff fundus" case, with negative Wassermann.

TABLE 3. ADRENAL DYSFUNCTION. 5 CASES IN 155 B SERIES.

Positive eye findings shown in 4 cases:

Enlarged Blind Spots	4 cases
Contracted Color Fields	1 case
Yellow Discs	1 case
Other Disc Changes	1 case

Percentage of positive eye findings = 80.

The blind spot enlargements were all slight to moderate in degree. The color field changes, noted in one case, was somewhat irregular and more marked in the right eye. The case showing yellow, tallow colored discs had normal acuity of vision, and normal fields except slight bilaterally (temporal) enlarged blind spots.

TABLE 4. PITUITARY DYSFUNCTION. 98 CASES IN 155 B SERIES.

Positive eye findings shown in 77 cases:

Enlarged Blind Spots	67 cases
Yellow Discs	30 cases
Other Disc Changes	6 cases

Form-Color Field Changes	23 cases
Scotomas	2 cases
Vascular-Pigment Changes	4 cases
Tumors	2 cases

Percentage of positive eye findings = 78.5.

This group contains the major point of interest and demands further study. Blind spots were mostly uniformly enlarged binocularly, ranging from two to five degrees radial increase. The discs varied from *distinctly yellowish* to *yellow tallow color,* without loss of substance. A few cases showed blurred disc margins or indistinct discs. Field contractions were mostly concentric, some within the blind spot area. Two cases showed monocular macular lesions, eliciting small central scotomas. One hypophyseal tumor case exhibited a blind left eye and a temporal hemianopsia in the right. Another pituitary case, with brain tumor, showed papilledemas of five diopters right and seven diopters left. Autopsy disclosed a neurofibroma, left cerebellopontine angle.

Several cases have been tested after treatment, and no. 71, (Fig. 2) illustrates a reduction in blind spot size after eight months pituitary gland therapy. Case no. 70[7], is of special interest, an almost totally lost left field having recovered to normal in one year, with a reduction of the right blind spot, under endocrine therapy for pituitary dysfunction with anterior lobe under- and posterior lobe overaction.

TABLE 5. SUMMARY OF 155 B SERIES AND 125 S SERIES TOTAL 280 ENDOCRINE CASES

	Thyroid	Gonad	Adrenal	Pituitary	Polygland. and Unclassified	Total
Positive Eye Findings	11	20	4	131	14	180
Normal Eye Findings	16	38	1	35	10	100
Total Cases	27	58	5	166	24	280

Illustrative Cases.

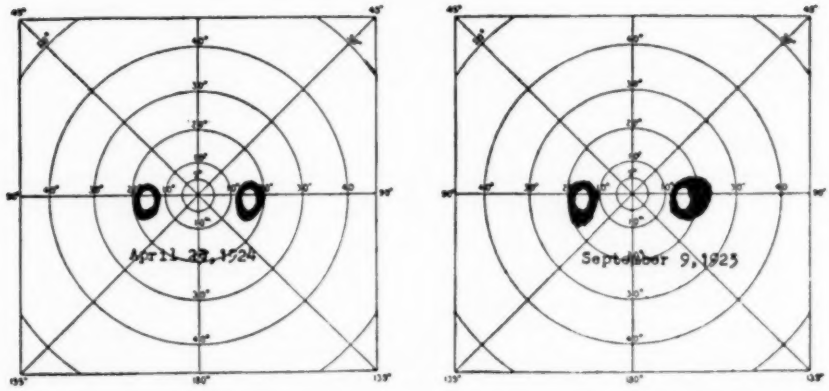

Fig. 1. Case No. B-71. Miss E., age 49. Pituitary Dysfunction. Ant. lobe underaction: Post. lobe overaction. Obesity. Neuropathic keratitis right. Yellow discs. Increased density of hypophysis and erosion of posterior clinoids. Treated. Reduction of blind spots in 7½ months.

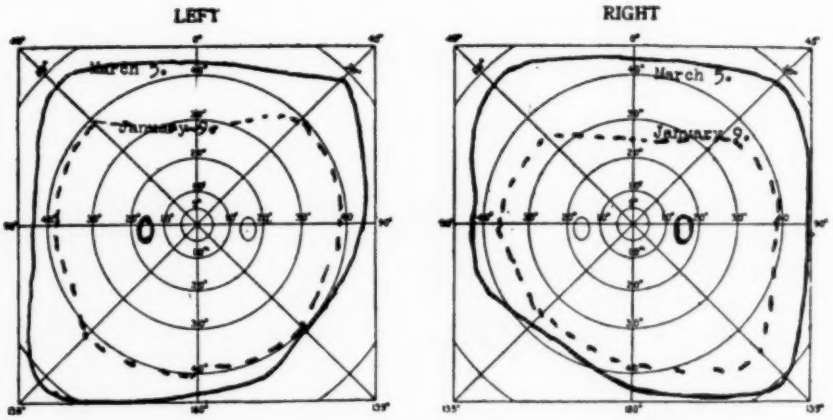

Fig. 2. Case No. B-110. Mrs. S., age 53. Thyroid hypofunction, treated. Form fields at three months interval.

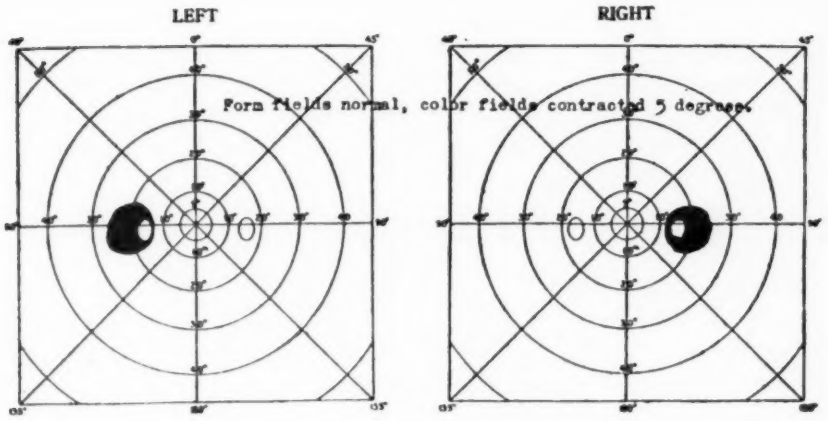

Fig. 3. Case No. B-119. Miss O., age 10. Pituitary dysfunction. Post. lobe underaction. Marked obesity; weight 195 pounds. Sella 8 mm. x 3 mm.

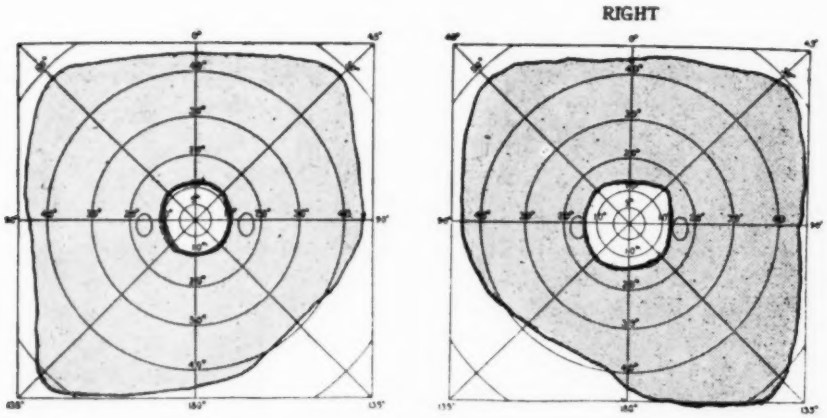

Fig. 4. Case No. B-212. Master O., age 15. Pituitary dysfunction. Ant. lobe underaction; Post. lobe overaction. Height 5 ft. 8. Large abdomen (ascites). Skull 9 mm. thick, sella small.

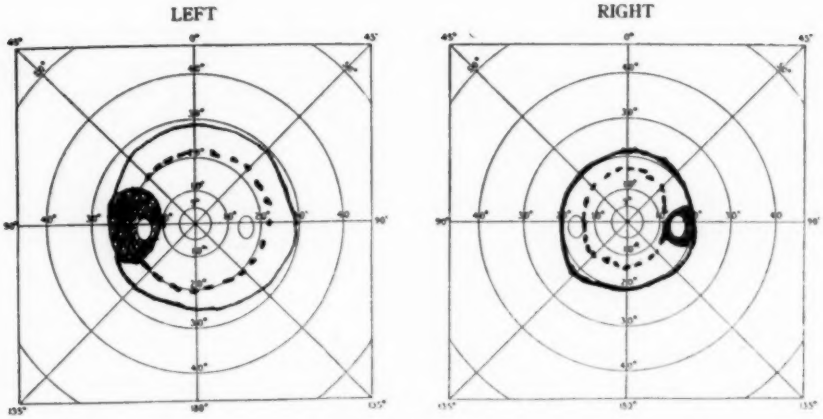

Fig. 5. Case No. B-127. Miss S., age 18. Pituitary dysfunction. Ant. lobe underaction; Post. lobe overaction. Severe anterior headaches, fatigue, obesity, nervousness. Sella normal. Discs yellow (Form field ——. Red field - - - -).

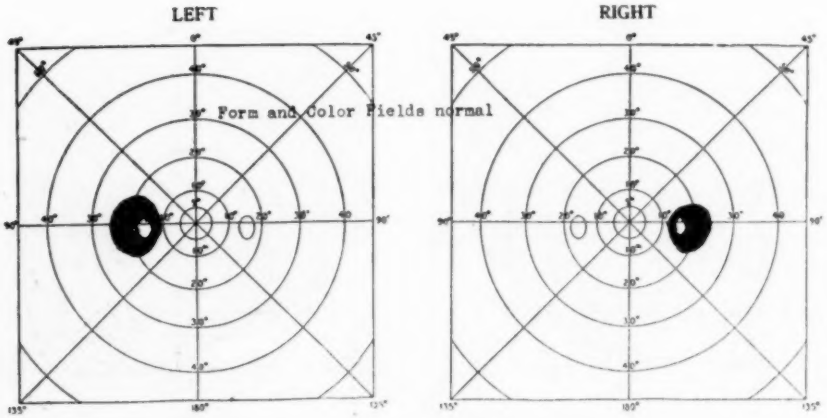

Fig. 6. Case No. B-270. Mrs. C., age 36. Pituitary dysfunction. Ant. lobe underaction; Post. lobe overaction. Yellow discs. Sella negative.

COMMENT: Only cases from the B Series were analyzed in tables 1-4, because they had more complete and intense study by the entire staff of the Clinic; however, the precentage ran about the same in both B and S Series, so far as the eye findings are concerned. It is keenly appreciated that deciding whether a disc is yellow, taking into consideration the many factors entering into such a decision, is a point requiring sane judgment; even so, exception may be taken to the observer's opinion in this connection.

Because of the anatomic relations, the pituitary cases are of greater interest, and seem to offer more reason for the pathologic entities elicited in this study. Why blind spots should be enlarged more frequently than temporal fields should contract, is not explained. Radiographs were taken of all cases, but their interpretation requires great care in relation to the individual, because of variations in age, sex, stature, etc. Correlations are under investigation at the present time, and will be reported in a later paper. Further study is also under way with the pituitary group, and includes more exact field change measurements by the use of the Feree perimeter, continued careful checking of blind spots on the stereocampimeter, analysis of radiographs of the sella, optic canals, and postnasal sinuses, and careful rhinoscopic examinations. Analysis of the findings thus obtained, with the subjective and objective symptoms manifested by the patient, in conjunction with the detailed vital function tests, may offer an explanation of the incidence of the various eye findings.

Previous work in this field by Erdheim and Stumme[13], Cushing[14] on fields in pituitary disease; Findlay[15], and Lancaster and Carvill[16], who found bitemporal cutting in pregnancy; by Benedict[17] who, among other factors in pituitary disease, described the yellow discs; by Hill[18], who reports the occurrence of enlarged blind spots in hypopituitarism, has been consulted.

The author wishes to express to Dr. Luther Peter his appreciation for kindly advice and encouragement in working out a large series of cases, and especially to thank Dr. A. W. Rowe and the staff of the Evans for the privilege of participating in this interesting work.

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DISCUSSION.

DR. LUTHER C. PETER, Philadelphia: Field disturbances and blind spot enlargements have been noted from time to time in endocrine dysfunction. Bizarre types of field changes are known to be present in a considerable number of cases, especially those primarily of pituitary origin. These changes have been confirmed by the findings in the series analyzed by Dr. Rowland. The most important observation, however, in this series is that of the enlargement of the blind spot in 69 per cent of the cases of pituitary dysfunction, 80 per cent of the adrenal cases, 44 per cent of the thyroid, and 36 per cent of the gonads.

The technic which Dr. Rowland employed is in keeping with the more exact methods now advocated as necessary to be of real scientific value. All doubtful cases were carefully checked on the Lloyd slate, and only those showing at least two degrees of enlargement, were recorded as pathologic enlargements. It might be well for future study to subject each case so reported to a study on a tangent plane of a one meter radius with a minute test object. This would tend to even greater refinement than the careful method reported in the paper. With all due allowance, however, for any errors which might have crept into a series of 280 cases, the number of enlargements of the blind spot in the series is of sufficient magnitude to claim our attention.

One of the interesting features is the high percentage of enlargements in the thyroid, gonad and adrenal cases. Field changes in cases of primary pituitary dysfunction are easily understood. Enlargements of the blind spots, as well as other field disturbances in dysfunction of the other glands mentioned, can be interpreted only as indicative of pituitary disturbance, caused secondarily by dysfunction of the other glands. It is this very intimate relation of the endocrine system which renders a study of endocrinology so elusive. The literature is rich in illustrative cases which establish this intimate relationship. Most conspicuous are the cases of pituitary disturbance found in pregnancy, in menstrual disturbances, in early adolescence, after double oophorectomy, and in thyroid disturbance. The relation of adrenal dysfunction to pituitary disturbance is

not so patent, although four of the five cases reported by Dr. Rowland show this relation.

The reasons for enlargement of the blind spot in endocrine dysfunction can only be surmised. Other field disturbances, in all probability, are due to hyperplasia of the pituitary, and to the resulting edema which surrounds the chiasm and the optic nerves. Possibly, as Traquair and de Schweinitz have suggested, a toxin from the surrounding edema, or a specific toxin elaborated by pituitary cells, finding entrance to the subarachnoid spaces of the chiasm and nerves, may explain a selective action when the papillomacular bundle is involved, as shown by the accompanying central scomoma. It would seem, however, that the pressure theory can account for most of the bizarre field disturbances encountered. Pressure may also cause enlargement of the blind spots. This seems the more likely, because choked disc is observed as an occasional symptom in pituitary disease. It seems to me more likely, however, that the many cases of blind spot enlargement can be brought about as in sphenoid and posterior ethmoid disease. It is as easy to believe that toxins from the inflammatory exudate surrounding a cystic hypophysis, or a specific toxin elaborated by the pituitary itself can gain entrance to the subarachnoid spaces and involve the peripheral fibers of the optic nerve, as that similar toxins can reach these spaces from diseased ethmoids and sphenoids. The peripheral position of the peripapillary fibers in the optic nerve is maintained back to the chiasm. Furthermore, by this same method, one can explain the presence of a central scotoma rather than by a selective tendency of the toxins to invade the papillomacular bundle. The macular fibers reach the temporal periphery of the optic nerve as it leaves the eyeball and therefore are open to invasion of any toxic process which may reach the subarachnoid spaces in the anterior part of the nerve.

All this is but theory, which can not be confirmed by pathologic study. The fact remains, that whether blind spot enlargement is attributed to pressure or to toxins within the subarachnoid spaces, it is present in a large number of cases of endocrine dysfunction.

The studies which Dr. Rowland has made are to be commended. Only by the accumulation of such data as he presented in his paper can we hope to master the great bearing which endocrine dysfunction may have upon the many symptoms and signs of disease which are as yet obscure.

Dr. Wm. L. Benedict, Rochester, Minnesota: I want to congratulate Dr. Rowland on the persistence with which he has carried out this work. I think his figures show the value of doing routine ophthalmoscope examination in cases that are under care in institutions where the examination can be controlled. I remember being in Boston when Dr. Rowland was carrying an ophthalmoscope around through the wards and looking at these cases. He has opened up some fields of study, which if taken up and worked with controls, may lead to something quite valuable.

It is news to me that there are characteristic changes in the visual fields or in the fundus among people who have been subject to dysfunction of testicles, ovaries, adrenals or thyroid. As far as our own investigations have gone, we have found nothing characteristic about their visual fields; and in thyroid cases, only those changes which might be expected to result from general metabolic changes as a result of thyroidism

have been found. In hypothyroidism, there is an associated change in the fundus which has sometimes been mistaken for the pallor of the disc that occurs in chiasmal lesions. I think, if we compare the general color of the fundus with the color of the disc, we will find the paleness of the disc is not so striking as in cases of chiasmal lesions. We have described the waxy pallor of the disc which is not accompanied by loss of substance.

I feel that our classification of atrophy must be largely ophthalmoscopic, because if we depend upon some other basis such as etiology, we inject many unknown factors. If we depend entirely on the ophthalmoscopic appearance we have at least a specific fact that we can apply in every case. It applies most to these chiasmal lesions. If the disc has a waxy pallor, without loss of substance, it is quite probable that the optic is suffering from block due to pressure, and that atrophy of the nerve has not yet set in. Such an appearance of the disc indicates a vitality of the nerve that will permit of resumption of function when the pressure is removed. If, however, with the pallor of the disc there is a wide cup due to atrophy with loss of substance, the function will not be restored when the pressure is removed. Obviously, operations for restoration of vision in persons whose discs are found to be atrophic will fail.

This pallor should not be called discoloration of the disc. It is not discoloration as from pigment or some extraneous matter; it is simply an absence of the normal color of the disc, which leaves it in different shades of pink on down to pale, and is pallor, strictly speaking, not discoloration.

Dr. W. D. Rowland, Boston (closing): I think the study of endocrine conditions lead us into a rather large, a somewhat new and rather mysterious field, and it is only by very careful and continuous work that we probably will be able to elucidate definite facts that may be established.

In regard to Dr. Benedict's point on the color of the discs, I stand corrected. It was descriptive rather than an anatomic fact.

SOLID EDEMA OF THE LIDS.

S. JUDD BEACH, M.D.

PORTLAND, ME.

Solid Edema is the name used by Sir Jonathan Hutchinson to describe a hard thickening of the face, apparently induced by repeated attacks resembling erysipelas. Fifteen years ago, Pusey remarked that knowledge of it was not general, and cases repeatedly fail of recognition. This is still true and is to be deplored. The deformity, for which there is no certain cure, appears to be largely preventable by early treatment. Furthermore, there is reason to suspect that the disease is less rare than has been supposed, and, therefore, an unappreciated number of cases are now going on to disfigurement for want of recognition.

In the following cases, diagnosis proved easy when attention was focused on the course of the disease rather than on the appearance at any single examination. This is one important point in identification, and comes about from the fact that although the separate stages of solid edema simulate many other disturbances, their fluctuations are distinctive.

These cases also make intelligible the apparent contradictions in the testbook descriptions, and tend to reconcile disputes regarding symmetry, consistency, color, and nasal involvement.

COURSE.

The malady commonly begins with an initial attack resembling severe erysipelas or cellulitis. The skin is brawny, red and tender, and there is fever and prostration. This seizure, or first stage, subsides through a period of dusky swelling, pitting on pressure, which might be called the subacute or second stage. Frequently, the color then fades, and shrinking progresses until there remains only the pale hard thickening of the remission, or third stage, from which the name solid edema is derived. This course is subject to wide

variations. One phase or another may fail to occur, and the transitions may be almost imperceptible. Thus the subacute phase may be protracted by smouldering infection, or it may be punctuated by outbreaks of inflammation, which can be regarded as recurrences of the first stage. Yet, at another time, or in another patient, it may come on without a preceding acute attack. It may even be so mild as to show little or no color, distinguished only by becoming a little puffy and boggy. Occasionally, this pale, doughy condition persists for long periods.

Such changes are repeated at irregular intervals. Outbreaks may occur several times a month, or the disease may be quiescent for years. In general, it is noticeable that the onset, while varying widely in severity, usually tends towards mildness. Meantime, the permanent swelling, on the contrary, increases with each attack. From the foregoing, it is easy to see the origin of the text-book contradictions. One widely quoted account[1] that represents the edema as doughy, dusky red, and pitting on pressure, is portraying the subacute stage: while the equally authoritative[3] picture of a firm, colorless thickening which does not pit, applies to the remission.

Prevalence.

Such disagreements bewilder an investigator and may be partly responsible for the singular facility with which the disorder eludes diagnosis for years and even decades. Cases have actually been recounted with meticulous detail before national associations, without being recognized either by the essayist or his hearers. Hence color is given to the suspicion, just intimated, that other cases are about, the true nature of which is in a like manner being overlooked.

A still stronger reason for this belief is the promptness with which an observer, once acquainted with the disease, finds other cases to report. Not only is this true abroad, but over here Pusey[4], Weidler[5] and others have each collected a considerable group of cases within a comparatively short time.

Should it thus turn out that solid edema is not so unusual, it follows, as just said, that the unrecognized cases are in the course of developing that unsightly swelling for which the disease is named. Much of this disfigurement, in many in-

stances at least, we believe might be prevented by earlier diagnosis and treatment. At present, complete recovery is not anticipated. According to the most recent observations[6] a perceptible thickening can be expected to persist even after the progress of the disease has been checked by vaccines. Nevertheless, present experience leads to the belief that the permanent deformity will be least in those cases which are treated earliest.

Differential Diagnosis.

As has been said, the separate stages are apt to be mistaken for similar appearances due to other causes. The first is almost habitually called erysipelas or cellulitis; the second is confused with a variety of inflammations, from leprosy to angioneurotic edema; the third is said to be mistaken for lymphatic tumors, and other conditions as widely separated as myxedema and trichinosis. It is by the sequence of these changes that the diagnosis is established. Not that any fixed order is essential, but the transitions, as described, appear to be pathogonomonic.

This is illustrated by the following case reports, in which, evidence is presented in detail where it suggests a reasonable answer to the questions regarding the symmetry, color and consistency of the swelling, and concerning nasal infection. This last problem is of fundamental importance. There is a growing feeling that the exacerbations originate in the nose, and that even during the latent periods, the infection may be harbored there.

Report of Cases.

Case 1: The 11-year old daughter of an American farmer had been placed by the authorities in a Portland household. In Dec. 1923, a mild infection about the eyes had been called erysipelas. Doubtless this was the *initial attack*, as a few weeks later, on Jan. 18, 1924, she went to the Portland Dispensary for a "sore" in her left nostril. Examination revealed an infection of the nose, throat, and tonsils, for which a stock vaccine was administered over several months. By the next month, improvement had made it safe to removed the tonsils. The operation was performed by Dr. George O. Cummings.

Vaccines were continued, and the improvement progressed. On May 27, 1924, when I saw her in consultation with Dr. T. A. Foster, the appearance was typical of stage 2. There remained on the bridge of the nose only a suggestion of thickening, with a slight blush. The lids showed some swelling that pitted on pressure. The lower left was slightly discolored and marked by a brown spot, 1 x 2 cm. The corresponding nostril *showed one or two strings of secretion* bridging the septum to the lateral wall, and a little moist exudate on the middle tubinate. For this, a fresh coryza might have been partly responsible.

On June 20, 1924, three weeks later, the Dispensary record says she looked absolutely well. In a few days, however, she

Fig. 1.
Case 1. Becoming *Subacute*. 1, Swelling mostly one sided. 2, Dusky red. 3, Pitting on pressure.

developed her second attack. The attending physician noted nothing significant about the nose. Two days afterwards, though the subacute phase had begun, I was still able to get a culture from some *thin pus* on the swollen left turbinates, yielding a pure culture of staphylococcus albus. As seen in Fig. 1, the swelling was *asymmetric*. It was *dusky* and *pitted on pressure*. By the following week, it had subsided both on the cheek and in the nose. By comparing her picture taken during the subsequent remission with that of her brother, it could be seen that the bridge of her nose looked a trifle the thicker. Palpation showed a slight abnormal *symmetric* thickening. Aside from this rather dubious indication and the trifling *dry crusting*, nothing unusual could be seen. Since this time, autogenous vaccines have been given, and no acute attacks have followed.

The next case was similar, but much more advanced, with marked permanent swelling.

CASE 2: A girl, 13 years old, had had "blood poisoning" of the face in Italy about 1916. Succeeding exacerbations of varying severity had lead to increasing swelling of the nose and eyelids. In 1921, she had had careful laboratory tests by Dr. Mortimer Warren. These revealed, among other things, a white count of 12,000, with 63% of polymorphonuclears and 1% eosinophiles. No parasites appeared. The urine examination showed a trace of albumin and a few hyalin casts. The blood Wassermann was anticomplimentary. In 1922, she had had nega-

Fig. 2.
Case 2. *Remission.* 1, Swelling symmetric. 2, Pale. 3, Not pitting on pressure. (After severe unilateral attack.)

tive rhinologic and X-ray reports from Boston authorities, but in 1924, when I saw her in consultation with the late Dr. Addison S. Thayer, there was a *little crusting,* like that in case 1, about the vestibule of the left nostril and the adjacent septum, which recurred in spite of treatment. A similar condition of the right side disappeared under mercurochrome ointment. Her appearance at this time was typical of the remission. A white, unyielding, symmetric thickening over the bridge of the nose extended to all four lids, as shown in Fig. 2.

Many diagnoses, which had been offered by her different advisors, both here and in Italy, have been discussed in a previous

paper[2]. That the case was one of solid edema had not been suggested. This conclusion was confirmed by a flareup while under my observation, in January, 1924. It followed exposure to cold, a common experience first mentioned by Jonathan Hutchinson[7]. The swelling was *asymmetric*, almost closing the right eye. It was *angry red and smooth*, simulating erysipelas. Oddly enough, it was the opposite nostril that was affected, and nearly occluded by the engorged middle turbinate. Between the turbinate and the septum, there appeared a little thin pus, yielding a pure culture of staphylococcus aureus. The temperature, at first 104 degrees, dropped rapidly and the swelling passed off along the cheek and neck as was usual with her.

Her subsequent history has been uneventful, with no further outbreaks. Whether this is due to an autogenous vaccine prepared by Dr. Mortimer Warren, cannot be determined so soon, as much longer remissions often intervene with no treatment whatever.

Comment.

In these cases, no matter how fleeting and unreliable were the manifestations upon which the text-books would base a diagnosis, the changes were absolutely characteristic. Regardless of irregularities in the order and duration of the cycles, the nearer the remission approached, the paler, denser and more symmetric became the swelling.

Conversely, it was more boggy and darker just after the onset. While this evidence sustains Adam in his contention that Hutchinson is in error in regarding the symmetry as characteristic, it does not prove that the exacerbations must be unilateral. Presumably the writers are at variance only because they refer to a different stage of the same disease.

Etiology.

These observations contribute little that is new regarding the etiology. The regular accompaniment of nasal disturbance yielding pure cultures of staphylococci, together with absence of special indications of tuberculosis, elephantiasis or eczema, often assumed to be factors, is consistent with the hypothesis previously suggested[2], namely, that such patients have a peculiar susceptibility to some one of the common skin cocci, and

are unable wholly to throw off the infection. Analogous conditions are familiar in which infectious foci, dormant for long periods, flare up when the resistance drops. Such occur in apical tooth root abscesses and recurrent appendicitis.

Nasal Infection.

It does not necessarily follow, of course, that the nidus is in the nose, because a vaccine developed from the pharyngeal organisms proved beneficial. We now know that such an action need not be specific.

Nor is it proved that the nasal infection, when it occurs, is primary. On account of the paradoxic inflammation of the right eye and the contralateral left nostril in case 2, while the contiguous organs were hardly disturbed, the direct connection between the intranasal infection and that in the face is not perfectly clear, although some definite relation must be admitted. I had the opportunity of observing each of these patients during an acute exacerbation. Invariably, nasal engorgement with purulent exudate began simultaneously with the outbreak in the skin, and subsided during the remission.

Yet the fallacy is demonstrated of the contrary theory, that nasal infection can be dismissed as a factor unless evidence of it has been specifically remarked. After negative rhinologic reports at competent hands, each of the present cases nevertheless developed the nasal disturbance described. I cannot help leaning to the opinion that the slight dry crusting, which I found constantly during the latent periods, had also been present during those examinations, but was looked upon as the ordinary secretion commonly found in healthy subjects. It varied in extent, and at its minimum was only a thin pellicle, hardly noticeable on the edge of a turbinate or forward on the septum. When it became more extensive, it resembled the description given by Adam[9] and others of the exudate seen by them. Much of the time, it would not have been considered seriously as evidence of a nasal disorder.

Much more general pharyngeal disturbance has been reported. At times, it almost overshadows the skin inflammation. An extreme example, if we accept Adam's very probable interpretation, was that narrated by Halsted[10]. His account mentions dry crusting over the middle turbinate and both sides of the septum, with a little pus in the right middle

meatus. In this case, however, a peritonsillar infection of four months standing had preceded, leading to thickening of the soft palate and over the arytenoids. The duration of this widespread pharyngeal inflammation for five years before the first outbreak of erysipelas, was cited in favor of the opinion that the disease starts in the nose. There followed 5 or 6 of these acute skin attacks, after the very first of which it was noted that they differed from ordinary erysipelas, in that the thickening of the skin persisted after the acute disease had subsided. Except for the more magnificent scale, the resemblance to the present cases is obvious.

One feature that seems to me highly significant, is that the nasal secretion was preponderantly one sided. Not that this is likely to be invariable in solid edema, as there is no obvious reason why the same process could not attack both sides simultaneously. Nor need it be mentioned that many local nasal lesions, such as sinus diseases and diphtheria, can also be unilateral. Yet it might help in differential diagnosis to bear in mind, that the nasal crusting of ordinary catarrh is bilateral, whereas an obstinate condition of like appearance occurring in solid edema has often been mainly unilateral.

Nomenclature.

As might be anticipated with these protean changes, there is no monotony in nomenclature. The very multiplicity of names shows that none are wholly satisfactory. Some like elephantiasis nostras (Sabouraud), lymphangitis facei (Mackenzie) and persistent lymphatic edema, apply only to one stage of the disease. In checking them off, it seems hardly possible that solid edema can be identical with so many different conditions, some of which, like erythema persistans and erysipelas persistans facei, have been carefully differentiated[11]. The mortality of the first attack of the latter disease is placed at 50%. It is not clear how it could be known that a patient dying in the initial seizure would have developed solid edema. In our first case, the onset was mild.

Falta speaks of "stabile erysipeloid edema." Such a name has merit, but it is not the erysipeloid phase that is "stabile." Stability is furthermore common to many facial swellings. It is the changes that are here characteristic. Very likely the knowledge simply of a recurrent erysipelas with chronic thick-

ening would suggest the diagnosis. While this description is not ideal as a title for the disease, on account of its length and perhaps the unwarranted implication in "erysipeloid," it may serve to suggest something more apt.

Treatment.

The object of diagnosis is to procure prompt treatment. The most satisfactory results reported have followed intranasal measures[9] and the use of vaccines[12-8]. Owing to the protracted remissions, which occur without any care, no conclusions can be drawn from the cases reported here until several more years have elapsed. On purely theoretic grounds, I still incline[2] to the latter expedient.

Summary.

Solid edema of the lids usually goes on to permanent deformity for want of early diagnosis and treatment. This is unfortunate, especially as the disease may be commoner than has been supposed. The wide variations at different stages in symmetry, consistency and color, have lead to contradictory text-book descriptions, which account in part for lack of recognition. The nomenclature is also confusing.

Diagnosis proved easy when attention was focussed on the course of the disease rather than on the appearance at any one examination. Neither the erysipelatoid outbreak, the period of decline, nor the permanent hard swelling is distinctive, but the fluctuations have a definite relationship.

Many attribute the disease to chronic nasal infection. This may be slight and unilateral.

The disease has been arrested both by intranasal surgery and by vaccines.

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DISCUSSION.

Dr. Arthur J. Bedell, Albany, New York: Dr. Beach in this, his second communication on solid edema, has summarized his findings, accurately defined the clinical course of the disease and suggested treatment.

In going over the literature, it is noted that there are several cases not included in his summary. For example, Shoemaker had two cases characterized by conjunctival irritation, lacrimation and lid edema. These followed thyroid removal and were cured by endocrine treatment. Fox reported a case in which for thirteen years there had been recurring swellings of lids, ears and face. The attacks would last from three to ten days and suggested erysipelas. The only etiologic factor was the reduced breathing space, the result of a broken nose. Rubio had a patient in whom the onset was insidious. There was no lacrimation or conjunctivitis. Other cases might be recalled, which, although not positively proven, are sufficiently suggestive to be included in this group.

Most clinicians agree that the diagnosis can be positively established only after the symptoms have persisted for a long time. The treatment has not been very satisfactory. An associated nasal condition has been frequently observed and it is, therefore, recommended that Ziegler's advice to treat the nasal conditions be followed. As a result of this exposition, these cases will be diagnosed earlier and treatment will probably be more efficacious.

Dr. Charles A. Bahn, New Orleans: We are indebted to Dr. Beach for calling to our attention a very interesting condition, which is apparently a type of reaction caused by several different factors, and is not a specific entity caused by one organism alone. I recall a somewhat similar case due to lues.

Fundamentally, the condition results from an alteration in the quantity or quality of the lymphatic supply to the part, with or without changes in the fixed cells, which affect the natural drainage outlets. In other words, there is either too much fluid carried to the part, or too little taken away. As time goes on, fibroblasts are deposited in the tissue and cicatricial changes begin. It is quite probable that developmental factors of the part predispose it to physical or chemical actions of various sorts, which result in the unusual reaction which Dr. Beach has called to our attention.

Dr. S. Judd Beach, Portland, Maine (closing): In closing, I wish to correct the impression that the diagnosis is necessarily delayed until the disease becomes advanced. When the entire paper is printed, it will be seen that in the first case, on the contrary, the pediatrist made the diagnosis shortly after the onset from the characteristics mentioned.

In favor of Dr. Bahn's position is the observation that these cases have always been particularly susceptible to exacerbations after chilling. The attack that I saw in the second case came on after the child was turned out of school into a blizzard.

TRANSACTIONS

OF THE

THIRTIETH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OTO-LARYNGOLOGIC DIVISION

NEW METHODS AND APPARATUS FOR TESTING HEARING.*

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NEW YORK CITY.

During the past four or five years, a new interest has been taken in methods of testing hearing. The making of actual measurements is certainly the basis for progress in understanding the many difficult problems concerning hearing. Since the advent of the vacuum tube and the stimulated interest in acoustics brought about by radio broadcasting, new tools have become available, which, when given the proper form, are valuable to the otologists as measuring instruments. It is the purpose of this paper to describe some of these new tools, and to show their relation to the the older methods of testing hearing.

The kinds of hearing tests which are needed may be classified into four groups according to their purpose as follows:

1. *Industrial,* or those made for determining the fitness of a candidate for employment. In certain types of work, it is particularly important that a prospective employee meet a definite requirement for acuity of hearing. Tests made in the army and navy for various branches of service are conspicuous examples of this kind of test.
2. *Educational,* or those made for determining the degree of hearing of school children, both in the public schools and in the schools for the deaf, for the special purpose of determining the proper methods to be used in their education.
3. *Clinical,* or those made for assisting the doctor to make a proper diagnosis of the cause of deafness.
4. *Research,* or those made to determine new facts about both normal and abnormal hearing.

It is evident that different methods are appropriate for these different purposes. Unless these various purposes are kept in mind, there is apt to be confusion when discussing the merits of various methods of testing hearing.

*From Bell Telephone Laboratories, Inc.

It is highly desirable that a single scale be used for representing the degree of hearing, which is independent of the method used, and which has a general application to all the purposes mentioned above. Therefore, before describing the apparatus developed for making hearing tests for these various purposes, I desire to describe the scale of hearing which is now being used by a number of otologists, which may be used for all these purposes, and show how the results obtained from the commonly made voice test, watch tick, acoumeter, coin click, and tuning fork tests can be converted into hearing loss units on this scale. This scale for measuring hearing was first described in a paper given before the Franklin Institute in September, 1923. It is the one used on the audiometers manufactured by the Western Electric Company. However, the use of this scale is in no way dependent on the use of audiometers of this kind. The units have been found to be very practical for describing the amount of hearing, since one of the units corresponds to a fractional change in the intensity of a sound, which is approximately the smallest that the average ear can detect. For example, when one changes the intensity of his voice from that corresponding to the musical notation PP (pianissimo) to that corresponding to the musical notation MF (mezzo forte), the loudness change is about 15 of these units. There are about 15 loudness steps in this range that the ear can recognize as being different. The range of intensities from the threshold of audition to the maximum which the ear can tolerate is approximately 130 of these units, depending upon the kind of sound. The hearing loss is defined accurately by the equation:

$$\text{H.L.} = 10 \log. \frac{I}{I_0}$$

where I and I_0 are the intensities necessary for the sound to be just audible to the person being tested and to a person of normal hearing, respectively.

It is not necessary, however, to remember this formula, or even to understand its significance, to be able to use in a practical way the scale of hearing just described.

In a paper presented before the American Triological Society by Fowler and Wegel (Audiometric Methods and Their Applications, May, 1922), a hearing scale was proposed which has been objected to by some otologists, because it is dependent upon the threshold of feeling as well as the threshold of

hearing. On this scale, the per cent hearing loss is the number of sensation units from the normal to the threshold for the patient, divided by the number of sensation units from the normal to the feeling point for a person of normal hearing. It is undoubtedly the best answer to the practical question as to what is the per cent hearing loss, and is very useful in expressing general results. It is particularly useful for describing to the patient his degree of hearing. However, for an accurate expression of the degree of hearing loss, it seems desirable to express results in terms of sensation units rather than per cent hearing loss.

The hearing loss expressed in sensation units can be converted into per cent hearing loss by multiplying the former by a factor, K, which varies with the type of sound used in the test. It is obvious from the definitions given above that this factor is equal to 100 divided by the number of sensation units between the threshold of hearing and the threshold of feeling for the particular sound used. Experiments made in the Bell Telephone Laboratories have indicated that for speech this factor is .83; for the test tone of the 3-A audiometer, it is equal to 1.0; for a watch tick, coin click, or acoumeter, it is approximately 1.5. For pure tones, the value of K is dependent upon the pitch, and is given in Table I. The question marks after 32 and 48 indicate that these values are not accurately determined. By means of this table, the hearing loss in sensation units can readily be converted into per cent hearing loss. For example, if a test indicates that a person's hearing is 50 units down from normal, his per cent hearing loss is 50 x .83% or 41.5%.

TABLE I.

FACTORS FOR CONVERTING HEARING LOSS EXPRESSED IN SENSATION UNITS INTO PER CENT HEARING LOSS.

Vibration Frequency	K	Vibration Frequency	K
32.....	3.3 (?)	800	.77
48.....	2.2 (?)	1024	.76
64.....	1.5	1600	.77
100.....	1.28	2048	.77
128.....	1.09	3200	.81
200.....	.96	4096	.86
256.....	.91	6400	.94
400.....	.83	8192	1.22
512.....	.79	12800	1.42

Speech or 4-A Audiometer..... .83
Test Tone (3-A Audiometer).....1.00
Watch Tick or Acoumeter.....1.5

When speech tests, as ordinarily made by otologists, are made in rooms with highly reflecting walls or in a place where there is noise, it is impossible to reduce the results so that they may be compared with other results, because of so many uncertain factors. However, if the tests are made in a room which is heavily damped and quiet, the distance that numbers can be interpreted correctly is definitely related to the hearing loss on this new scale. In Table II are given numbers which show this relationship for four different intensities of voice. The derivation of these figures are based on some experimental data obtained in the laboratory, and from the inverse square law relating to the intensity of sound and the distance from the source.

TABLE II.

ROOM HEAVILY DAMPED AND FREE FROM NOISE.
Hearing Loss in Sensation Units.

Distance In Feet	Average Whisper	Loud Whisper or PP Voice	MF Voice	FF Voice
⅛	50	65	80	95
¼	44	59	74	89
½	38	53	68	83
1	32	47	62	77
2	26	41	56	71
4	20	35	50	65
8	14	29	44	59
16	8	23	38	53
32	2	17	32	47
40	0			
64		11	26	41
128		5	20	35
226		0		
248			14	29
496			8	23
992			2	17
1280			0	
1984				11
3968				5
1⅓ miles				0

The derivation of these numbers is more fully explained in a paper recently published in the Laryngoscope of July, 1925.

Only very approximate results can be obtained by such speech tests, since it is very difficult to determine the particular type of voice one uses in calling numbers. It is interesting to know, that when one is using a very loud voice corresponding to shouting, one can hear and interpret called numbers at a distance of 1⅓ miles provided, of course, that everything is perfectly quiet. A person with normal hearing can also hear and interpret numbers which are whispered at a distance of 40 feet, when everything is perfectly quiet. For example, if one is using an average tone of voice in a room which is heavily damped and free from noise, and finds that the patient can interpret his numbers at a distance of 4 feet, the hearing loss would be 50 sensation units. In most rooms in large cities, unless precaution has been taken to eliminate the noise, one will obtain distances corresponding to between 20 and 30 units loss in hearing for a person having normal hearing. It is also seen from the figures in Table II, that it is not practical to determine the loudness of one's voice by noting the distance that the normal ear can hear, since these distances are all too large to be obtained in an ordinary sized room. If distances smaller than these are obtained, it indicates that the noise has not been excluded.

This same method can be used for reducing watch tick or acoumeter results to the common scale, provided the intensity of the watch tick or acoumeter is known. A number of observations made with good grade watches indicated that a patient who could just hear a watch tick, which was held on the ear, has a 50% hearing loss in the neighborhood of 2,000 cycles.

In the standard method for determining the acuity of hearing at different pitches, use has been made of a series of tuning forks having various vibration rates through the audible range. The fork is given a standard blow of some sort to set it into vibration. It is then held as close to the ear as possible, preferably with the flat part of the prong directly facing the auditory meatus. The time, t, in seconds from the striking of the blow until the patient no longer hears the sound is observed. A comparison of this time to that, t_0, required for normal hearing gives a measure of the hearing by air conduction.

It is well known in mechanics, that after a tuning fork is

set into vibrations, the difference in the logarithms of the amplitudes of the sound emitted by the fork at two different times is proportional to the difference in the two corresponding times elapsed from the time of the initial blow.

Due to this relation and the way that hearing loss is defined on the new scale, it is easy to show that the hearing loss is equal to a constant, designated by Δ and called the damping constant, multiplied by the difference in time, t_0, for the normal ear, and t for the ear of the patient being tested. The constant factor for reducing the time difference to sensation units is dependent only upon the fork, and should be furnished by the manufacturer. For illustrating the kind of constants one might expect to obtain, the values of Δ for three groups of tuning forks are given in Table III. For convenience, the values of K from Table I are also given. The first group of forks are from a set used in the Bell Telephone Laboratories. The second set was loaned to us by Dr. E. P. Fowler. The last fork is a 500 cycle standard fork used by Dr. Douglas MacFarlan.

For example, when Dr. Fowler's 256 fork is used, the per cent hearing loss is given by:

Per Cent Hearing Loss $= 1.25\ (70 - t)$

where t is the air conduction time for the patient. It is evident from the formula that a person having more than 87½ per cent loss will not hear the fork at all.

TABLE III.

TYPICAL CONSTANTS FOR TUNING FORKS.

Rate in dv	Damping Constant Δ	Time, t_0 in seconds for Normal Air Conduction	Factor K for Reducing Hearing Loss to % Hearing
	BELL TELEPHONE LABORATORY FORKS.		
24	.30	75	4. (?)
48	1.9	51	2.2 (?)
64	1.61	41	1.5
100	1.75	30	1.28
200	.93	110	.96
400	.46	140	.83
500	.59	135	.81
800	.87	112	.77
1000	1.19	71	.76
1200	1.14	69	.76
1800	2.29	44	.77
2000	2.41	45	.77

DR. E. P. FOWLER'S FORKS.

128	1.08	65	1.09
256	1.38	70	.91
512	1.31	95	.79
1024	1.70	40	.76
2048	2.17	20	.77

DR. D. MACFARLAN'S STANDARD 500 CYCLE FORK.

500	2.0	55	.81

Hearing Loss $= \triangle (t_0 - t)$

Per Cent Hearing Loss $= K \triangle (t_0 - t)$

It is useful to notice, that if the hearing loss of the tester is known, then the hearing loss of the patient can be found as follows: Set the fork into vibration by any means, not necessarily using a standard blow. Hold the fork to the patient's ear in the standard fashion. Start the stop watch when the patient signals he no longer hears the tone. Hold the fork to your own ear in the same standard way. Stop the watch when you cease to hear the tone. Then the reading of the watch in seconds multiplied by the constant of the fork, $\triangle$, gives the difference in sensation units between your hearing loss and that of the patient.

This method has the advantage that the results are not dependent upon the initial blow given to the fork, but it has the disadvantage that it depends upon the hearing of the tester, and any noise in the room affects the hearing of the tester much more than that of the patient, provided the former is much more acute than the latter.

For example, suppose that you know that the tester's hearing loss is 20 SU at 1024 vibrations per second, and also, when using the technic described above and Dr. Fowler's 1024 fork, the time difference is found to be 25 seconds. Then, the hearing loss of the patient is

$1.70 \times 25 + 20 = 62.5.$

or his per cent hearing loss is

$62.5 \times .76 = 47.5\%.$

It would be a great advantage to those who use tuning forks, if the makers of such forks for otologic purposes would furnish values of the damping constants as well as the frequency of vibration. Then the results of tuning fork tests could be reduced to a common basis as indicated above.

In order to increase the speed and accuracy of making hearing tests which are equivalent to those which have been

described, and for the purposes outlined at the beginning of the paper, the Bell Telephone Laboratories have developed several pieces of apparatus which I wish to present for your consideration. For making speech tests, a phonograph type of audiometer has been developed. In this instrument, records which have been made by special electric processes are used for reproducing the speech sounds. This insures a definite volume of voice, so that accurate comparisons are possible. The recorded speech sounds are transformed into their electric equivalents by means of an electromagnetic reproducer. The electric waves thus created are carried by means of wires to a receiver which is held to the patient's ear. The intensity of the sound sent into the patient's ear is controlled by electric resistances in the electric circuit. This phonograph type audiometer is furnished in three different forms, according to the purpose for which it is to be used. For making a survey of the hearing of a large group, to find those who have a deficiency for hearing speech, it is furnished with the turntable unit, with special records upon which the recorded speech sounds continually decrease in intensity as the record revolves, and multiple units containing 8 receivers per unit. A master sheet is furnished, which gives the hearing loss corresponding to the intensity of each of the numbers on the record. In this form no batteries or attenuators are required. An instrument of this sort was tried out at one of the public schools in New York City, and it was found that with a single phonograph unit and three multiple units, that is 24 receivers, the pupils could be tested at the rate of 100 per hour. The entire school of about 1000 pupils was tested in three days, and a number determined for each pupil which represented his hearing ability for speech. (Demonstration of use of this instrument by connecting it to the public address system, and attenuating until a person in the front of the hall hears the numbers corresponding to normal.)

For purposes of testing more accurately the hearing of one individual at a time, an attenuator is interposed between the turntable unit and the receiver, and a record is used which gives a constant speech level. For use in schools for the deaf, or for testing patients who are very hard of hearing, an amplifier unit can be furnished to be interposed between the turntable unit and the attenuator box. (Demonstration of the use of this instrument with a 25-A amplifier.)

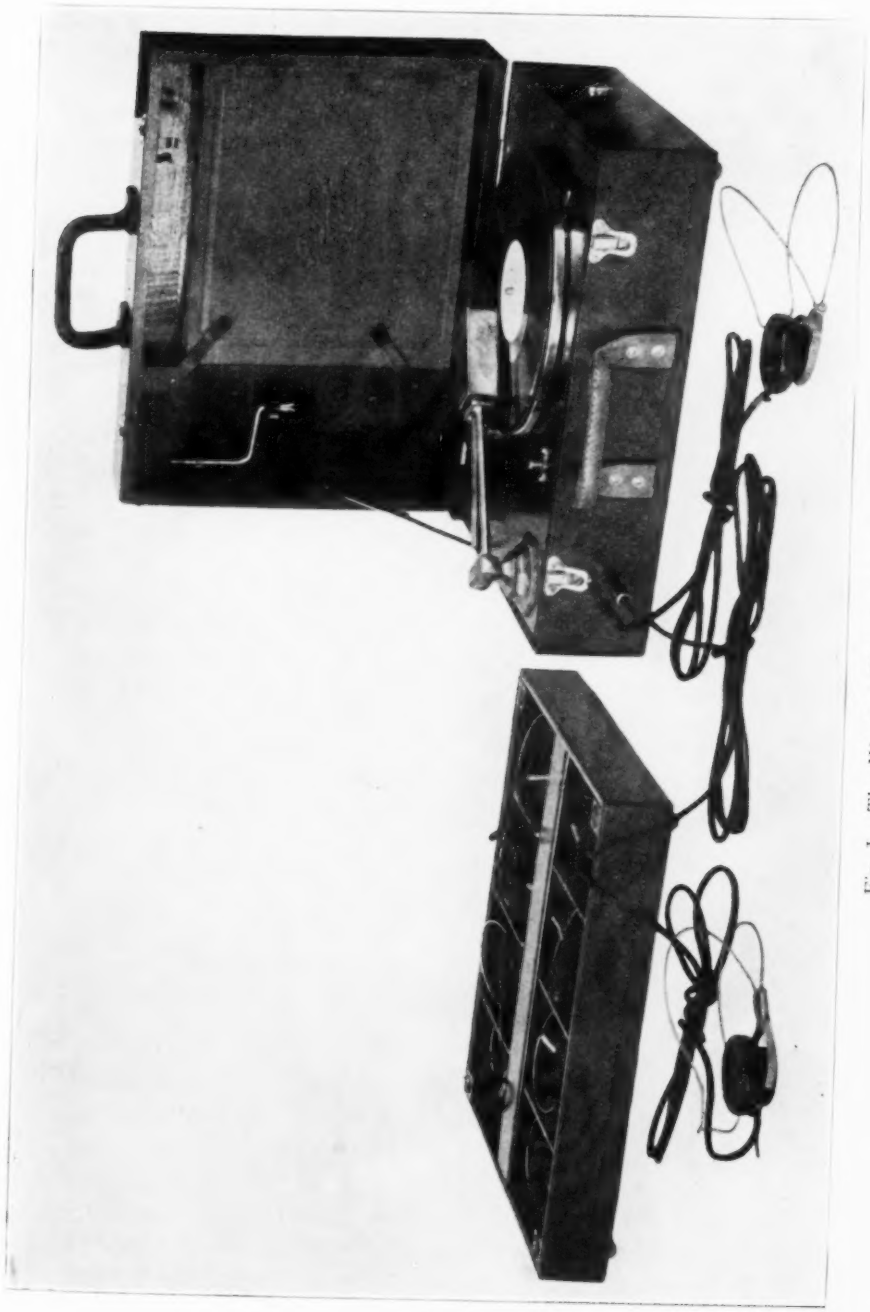


Fig. 1. The Western Electric 4-A Audiometer

This phonograph type of audiometer can also be used as an auxiliary to either the 1-A, 2-A, or 3-A audiometer. When used in this way the hearing loss can be read directly on the audiometer dial. (Demonstration of its use on both the 1-A and 2-A audiometer.) With this instrument, the speech intensities being delivered to the ear of the patient can be varied 10 billion fold, by simply turning the dial from one end of the scale to the other, corresponding to a distance variation of more than 100,000 times. This makes it possible to test all degrees of hearing from normal to totally deaf. Not only does it have this increased range over that obtainable in the ordinary speech test, but it has a definite intensity level for the speech, and is not subject to variations in the acoustic properties of the room.

The 3-A audiometer, which has been described before, has been developed to take the place of such tests as the watch tick tests, the acoumeter tests, or those designed to make a very quick test of the general hearing level. In this instrument, a tone having components throughout the entire speech frequency range is electrically generated and delivered to a receiver to be held on the ear of the patient. The volume of the tone is controlled by the same attenuator unit as used in the other audiometers. It reads directly either in sensation units loss or per cent hearing loss, since it was found for this tone that there were just 100 SU between the threshold of hearing and the threshold of feeling for the normal ear. This instrument has been found to be particularly useful in schools for the deaf. It enables the teachers to grade the degree of hearing of the child very quickly, which aids them in deciding upon the kind of methods to be used in teaching him. It is also useful in making a quick test of the hearing of large groups when they are tested one at a time. (Demonstration of the performance of the 3-A audiometer.)

This 3-A audiometer is also useful when making tests of bilateral deafness, for it can be used in place of the Barany's noise apparatus. For this purpose it is much superior to the latter device, because the volume can be placed at just the proper level to mask sufficiently the hearing in the good ear, without in any way interfering with the hearing in the bad ear. The proper level for doing this depends upon the relative difference in the hearing of the two ears. Those who believe an ear massage is of value may find an additional use for this instrument.

The apparatus which has been developed for replacing the tuning fork test is now quite well known. Several types of audiometers have been developed for this purpose. The two which have been developed by the Bell Telephone Laboratories and known as the 1-A and 2-A audiometers are here for demonstration. The 2-A audiometer has been designed for general practice, and for this reason great stress has been placed on the convenience and simplicity of its operation. Eight tones, spaced an octave apart, in the frequency range from 64 to

Fig. II. The Western Electric 3-A Audiometer.

8,192 vibrations per second, can be produced by throwing the appropriate keys on the top of the audiometer. The attenuator scale is calibrated, so that the hearing loss in sensation units is read directly from the scale when the dial is turned to the position corresponding to the threshold for the patient being tested. Audiogram blanks are furnished with the instrument, which makes it possible to show graphically the hearing loss at each frequency. Already a number of doctors have changed the form of these blanks to fit their particular need, but of course have retained the same scale.

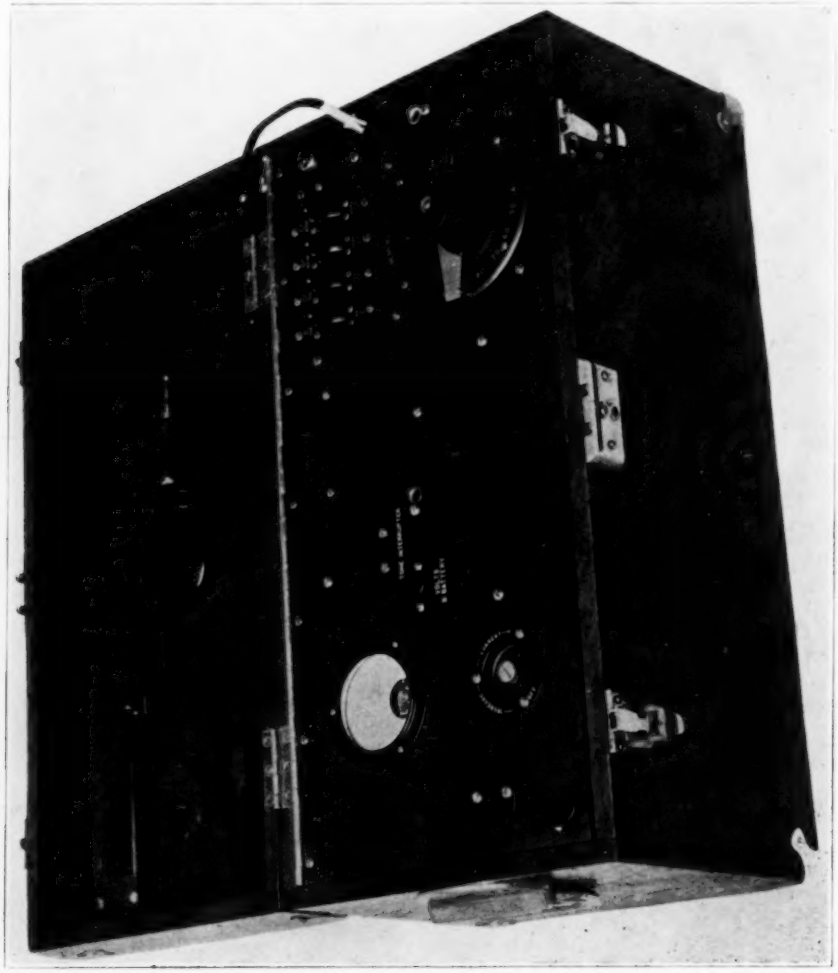


Fig. III. The Western Electric 2-A Audiometer.

The 1-A audiometer is similar in principle to the 2-A audiometer, but it has a much greater range in frequency and intensity. Both of these instruments are equipped with a jack, so that auxiliary equipment may be used with them. It has been our experience that the doctor frequently desires to talk to the patient either before or during the time that the pitch range test is being made. For this purpose, a talking set has been developed. By plugging the end of the cord of the talking set into the jack of the audiometer, it is possible to talk directly to the patient through the receiver which he is using during the test. By turning the attenuating dial, the volume of the speech thus delivered to the patient's ear can be varied in intensity from high values to the threshold of audibility. (Demonstration of the use of this instrument on both types of audiometer.) Also, as mentioned before, the phonograph audiometer may be used as an auxiliary to both the 1-A and 2-A audiometers, by plugging the cord from the turntable unit of the phonograph audiometer into the appropriate jack. Then, the speech waves coming from the phonograph record will be delivered directly to the receiver of the patient's ear. Its volume may be controlled by the attenuator dial. A mark on this dial can be determined, so that readings on the scale from this mark will give directly the patient's hearing loss for speech.

In schools for the deaf and other places where large intensities are required, and where the expense of a 1-A audiometer may not be justified, the intensities of the 2-A audiometr may be increased by adding the amplifier unit used in connection with the phonograph audiometer. This amplifier can be used with the 2-A audiometer by a very slight modification in the present type. (Demonstration the use of the 25-A amplifier with the 2-A audiometer.)

For research purposes, any or all of this apparatus might be used. It may be modified in a great variety of ways to suit the investigator. For example, three different types of tone range audiometers have been discussed before this society, in which a continuous variation in pitch was possible. It has been our opinion that except for special research work, this is not necessary or even desirable. However, for those who like the other features of the 1-A audiometer and still want a continuous variation in frequency, I will state that a 1-A Western Electric condenser box can be connected into the

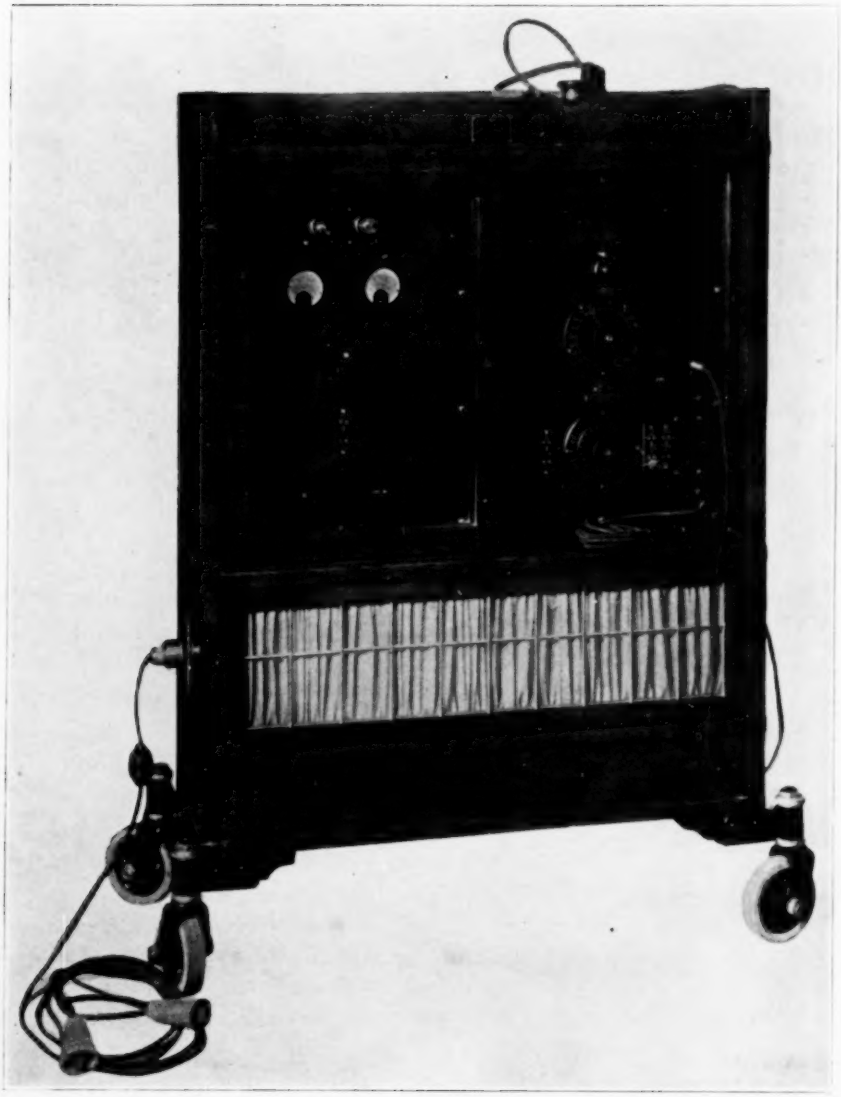


Fig. IV. The Western Electric 1-A Audiometer.

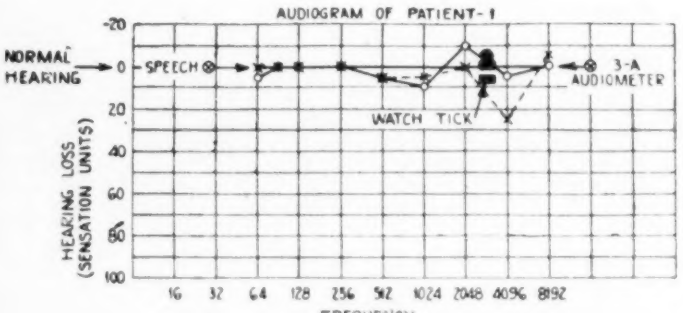
circuit, so that all frequencies besides those normally obtained can be produced. When the audiometer is thus converted, a calibration curve is required for interpreting the results.

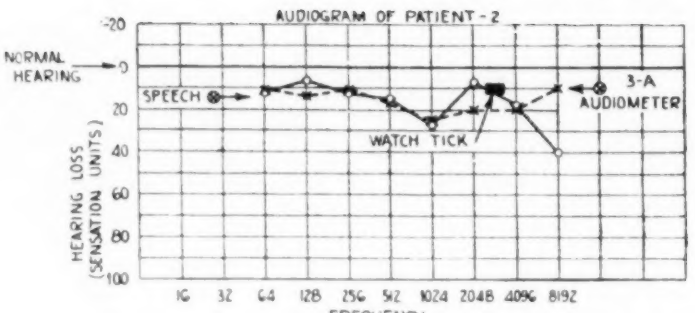
In order to show comparative results obtained by different methods of testing, Table IV is given. It shows the results of tests on the hearing of 10 persons by four different methods, namely, the phonograph audiometer, the 3-A audiometer, the standard speech test, and the watch tick test methods. Audiograms obtained by the 2-A and 1-A audiometers are given for these same people in Fig. V. On these audiogram charts, the hearing loss for speech and for the 3-A audiometer tone are shown at the left and right respectively. The black rectangle represents the watch tick results. A comparison of the results shown in columns III and IV shows that the phonograph audiometer gives results which are in good agreement with the reduced results from the standard speech tests. The differences are well within the observational error.

The watch tick results are just what one would expect from the amplitude frequency characteristic of this type of sound. It is interesting to note, that if patients 3 and 5 were given a watch tick test only, they would be considered to have the same amount of hearing; but further tests showed that patient 3 could hear speech at 20 feet, while patient 5 could hear it only 2 feet away. It is evident from the audiograms why this is true. Patient 5 hears the high frequencies very much better than the low frequencies. An examination of the various audiogram charts indicates that the 3-A audiometer really gives a good criterion of the general hearing level. However, results obtained by it are not definitely correlated to the hearing loss for speech, although there is a general agreement. A notable exception is the lack of agreement in the results obtained by patient 10. The 3-A gave 45 SU for the right and 60 SU for the left ear. The phonograph audiometer gave the hearing loss for speech as 70 SU for the right and 75 SU for the left ear. An examination of the audiogram for patient 10 shows why this should be. The important frequencies for speech interpretation, that is from 500 to 2000, are considerably below the general level, the low frequencies being the highest. This is also true for patients 8 and 9. For patient 5, the speech frequencies are on about the same level as the low frequencies, but very little loss is shown for the high frequencies. In this case it is probable that the 3-A audiometer

Table IV.

I	II		III	IV	V		VI	VII
	Distances for Interpreting Numbers			Hearing Loss	Distances for Watch Tick Test			Hearing Loss
Ear Number	Voice A $Y = 58$	Voice B $Y_o = 65$	Hearing Loss Cal. from II	Phonograph Audiometer	A (Y_o=10)	B (Y_o=15)	Hearing Loss Cal. from V	3-A Audiometer
1-R	800 ft. (cal.)	1800 (cal.)	0	0	40 in.	70 in.	0	1	0
1-L	800 ft. (cal.)	1800 (cal.)	0	0	20 in.	36 in.	6	5	0
2-R	140 ft. (cal.)	320 (cal.)	15	15	10 in.	31 in.	12	7	10
2-L	140 ft. (cal.)	320 (cal.)	15	15	15 in.	64 in.	8	2	10
3-R	20 ft.	20 ft.	32	?	30	3 in.	5 ½ in.	22	22	30
4-L	3 ft.	6 ft.	48	49	35	..	1 in.	..	39	35
5-L	1.7 ft.	3.7 ft.	53	54	45	2 in.	4.5 in.	26	23	40
5-R	1.8 ft.	3.7 ft.	52	54	50	3 in.	6.5 in.	22	21	45
6-L	2 ft.	3.5 ft.	52	54	50	..	contact		55	45
6-R	1.5 ft.	4 ft.	54	53	45	..	.5 in.		45	45
7-R	.9 ft.	2 ft.	57	59	50	..	contact		55	45
8-L	1.2 ft.	3.7 ft.	56	54	60	..	..			50
8-R	1.2 ft.	3.7 ft.	60	54	60	..	contact		55	50
4-R	No test				65	..	..			65
9-L	8 in.	13 in.	62	64	65	..	..			60
9-R	5 in.	8.5 in.	65	68	70	..	..			60
7-L	8 in.	15 in.	61	63	70	..	..			65
10-R	3.5 in.	9 in.	69	68	70	..	..			45
10-L	2 in.	6 in.	74	71	75	..	..			60
3-R	No test				90	..	..			80





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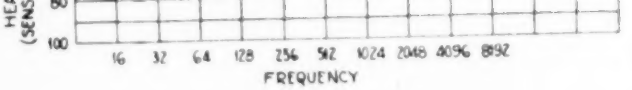
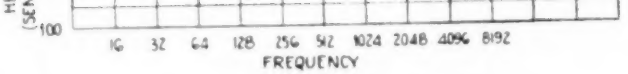
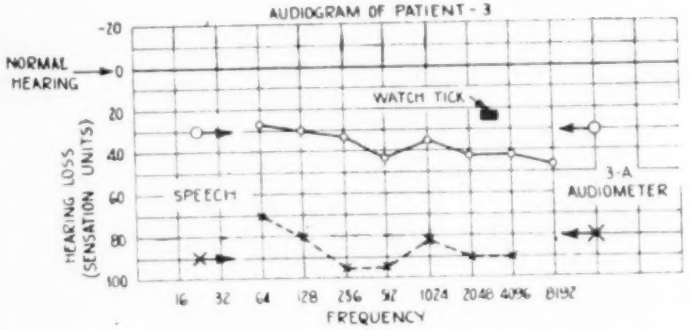
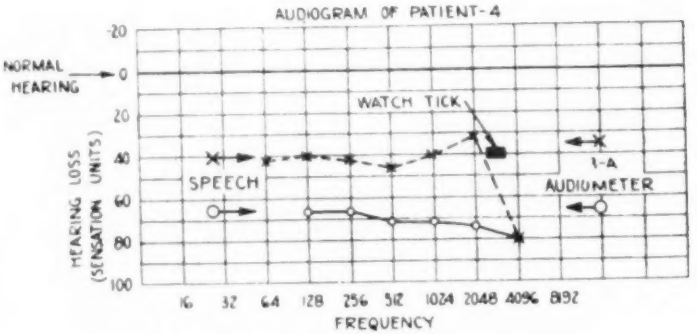
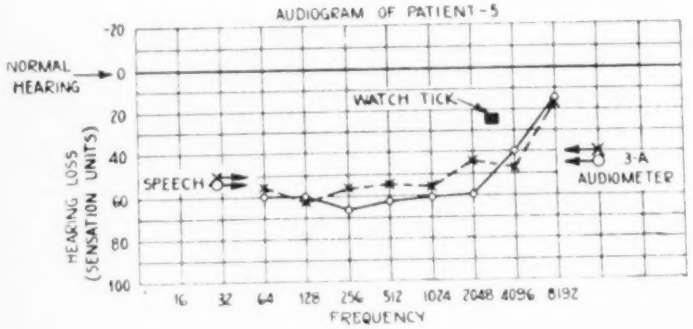
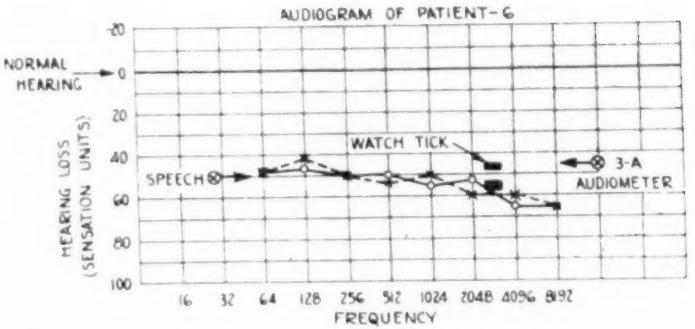

HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY

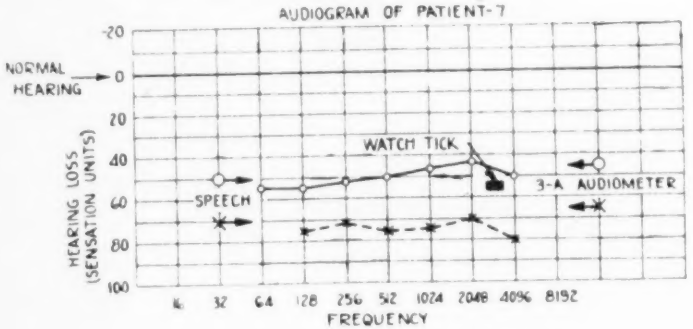

HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY

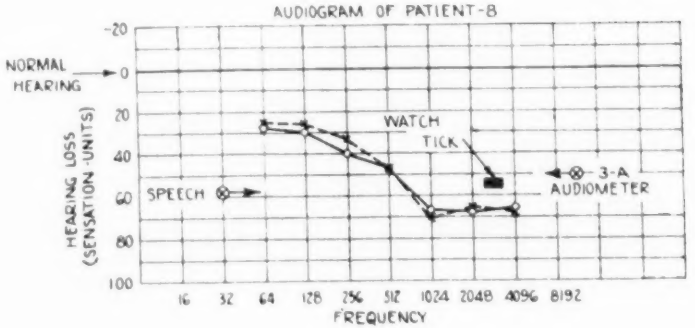

AUDIOGRAM OF PATIENT - 3
NORMAL HEARING
WATCH TICK
SPEECH
3-A AUDIOMETER
HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY

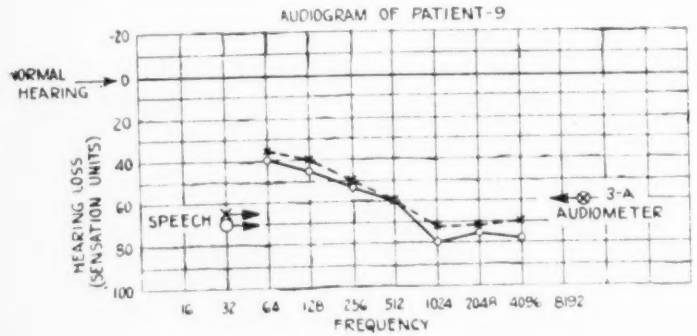

AUDIOGRAM OF PATIENT-4
NORMAL HEARING
WATCH TICK
SPEECH
1-A AUDIOMETER
HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY


AUDIOGRAM OF PATIENT-5
NORMAL HEARING
WATCH TICK
SPEECH
3-A AUDIOMETER
HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY


AUDIOGRAM OF PATIENT-6
NORMAL HEARING
WATCH TICK
SPEECH
3-A AUDIOMETER
HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY


AUDIOGRAM OF PATIENT-7
NORMAL HEARING
WATCH TICK
SPEECH
3-A AUDIOMETER
HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY


AUDIOGRAM OF PATIENT-8
NORMAL HEARING
WATCH TICK
SPEECH
3-A AUDIOMETER
HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY


AUDIOGRAM OF PATIENT-9
NORMAL HEARING
SPEECH
3-A AUDIOMETER
HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY
RIGHT EAR
LEFT EAR

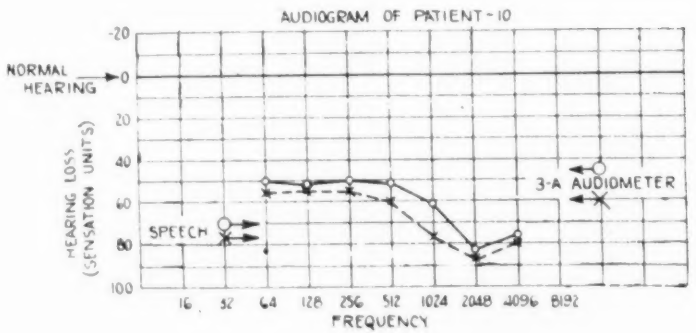

AUDIOGRAM OF PATIENT-10
NORMAL HEARING
SPEECH
3-A AUDIOMETER
HEARING LOSS (SENSATION UNITS)
-20
0
20
40
60
80
100
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY

Fig. V.

tone was heard because of components between 2000 and 8000 vibrations per second.

TABLE V.

HEARING LOSS FOR SPEECH.

Ear Number	Calculated From Audiograms	Observed Average
1—L	2	0
1—R	2	0
2—L	20	15
2—R	16	15
3—L	39	31
3—R	89	90
4—L	40	40
4—R	72	65
5—L	52	50
5—R	61	55
6—L	55	50
6—R	48	50
7—L	48	50
7—R	73	70
8—L	61	58
8—R	60	58
9—L	68	65
9—R	72	70
10—L	75	75
10—R	66	70

Our studies on speech and hearing have indicated that the important frequencies for interpreting speech are between 500 and 2000 vibrations per second. The simple method of taking an average of the hearing losses at 512, 1024, and 2048 for determining the hearing loss for speech, gives results in good agreement with observations on these 10 patients. In Table V is shown a comparison of results thus obtained. The observed values are averages of the figures given in columns III and IV of Table IV. The per cent hearing loss is obtained by multiplying the figures by .83. Until more accurate methods are devised, this simple procedure will be useful in calculating from the audiogram the per cent hearing loss for speech, which is the figure of greatest interest to the patient.

DISCUSSION.

DR. G. W. MACKENZIE, Philadelphia, Pa.: It will not be possible for one to cover in a discussion of ordinary length all the issues brought out in so lengthy a paper.

It is conceded that one ought to be able, with an audiometer such as that sponsored by Dr. Fletcher, to determine quite accurately the loss of hearing function in any given case. Furthermore, a technician could be taught how to make these tests quite as accurately as the otologist himself, and from the findings decide the fitness of the candidate for employment, or of children for one or another kind of school, as contended by Dr. Fletcher. So, too, can a technician be taught to examine the vision and ascertain quantitatively the visual acuity. But from this alone, he cannot make a diagnosis as to whether the lesion responsible for the visual loss is located in the optic media or in the perceptive apparatus—optic nerve and retina. The oculist can, however, with the aid of additional tests, for instance, those for color sense, perimetric and campimetric fields—obtain knowledge that will go a great way toward solving the problem of the location of a lesion. After a somewhat similar fashion, the otologists can, with the aid of certain functional hearing tests of a qualitative nature, tell the location of the lesion responsible for the loss of hearing. In fact, it is possible to tell by these tests not only the location of a single lesion, but in the case of mixed or combined lesions, the presence of each, and besides, which of the two lesions is responsible for the major part of the loss of hearing.

I do not wish to be understood as contending that it will never be possible to determine the location of the lesion responsible for impaired hearing with an audiometer. But there seems to be no promise of our being able to do so in the near future. In the first place, no one has, so far as I can learn, plotted any large number of audiograms and checked them up with fork test findings, in a manner at all convincing.

I wish to concur in the opinion expressed by Dr. Fletcher regarding the speech test, as ordinarily made, but with reservations. We cannot relegate the "speech test" to oblivion, since it serves us well in many ways, not the least of which is in the detection of malingering. Quoting the author: "In table two are given numbers, which show this relationship for four different intensities of voice." Since the whispered voice differs in different indivduals, I do not see how mathematic certainties can be expressed when we have such variable quantities to deal with. This same objection can be raised about the other three voices P P, M P, and F F voice. In the next paragraph, the essayist practically negates the value of Table II, when he says that only very approximate results can be obtained by such speech tests, since it is very difficult to determine the particular type of voice one uses in calling numbers. May I add, too, that the carrying power of the human voice is not dependent entirely upon the loudness of the voice, but also upon the clearness of the enunciation.

In the issue of the Laryngoscope called the Audiometer number, there is to be found, on page 528, the following paragraph, which I beg you to read carefully. It runs as follows:* "In case of impaired hearing due to obstruction to the external auditory canal, the chart curve will show

*The italics are mine and not the author's.

the greatest drop for low tones in case of simple type. The fineness of the audiometer record is such, that it *suggests* in some cases of this nature an irritable auditory nerve (*probably* an initial congestion incident to the *possible* complicating Eustachian tube factor). In such cases, the chart curve for high tones may also show a decided drop, which disappears promptly after removal of the offending obstruction in the canal, for the complicating Eustachian tube factor will recover spontaneously upon removal of the obstructing pathology in the external auditory canal."

To appreciate what the author is driving at needs more than one reading. It should be read several times. The use of such words as "suggests," "probably"" and "possible" all occur in the course of single sentence and is capable of but one interpretation, and that is a roundabout effort to show something that no otologist can possibly accept; to wit, that an external auditory canal obstruction produces an irritable auditory nerve. All normal auditory nerve endings are irritable, while the nerve itself merely acts as a conductor. This the author does not mean. We are left to assume that there is a pathologic condition present in the nerve, for in the second part of the same paragraph he says, that the sharp curve for high tones may also show a decided drop, which disappears promptly after removal of the offending obstruction, etc. And all this guess work is done to prove the *fineness* of the audiometer record. The word "fineness" is synonymous with the word preciseness or exactness. And this is from the pen of one who, in his summary on page 532, contends that the audiometer makes possible speedy and accurate tests, which eventually will prove of no uncertain value in medicolegal cases.

Now last, would any self respecting court accept as convincing the evidence presented in the paragraph on page 528, where such words as *suggest, probably* and *possible* all occur within one short sentence?

Upon comparing the audiograms of the different contributors to the symposium in the July number of the Laryngoscope, one finds a lack of uniformity of the curve for the same condition as observed by different contributors. Compare, for instance, the audiogram Fig. 8 on page 528 with the audiogram Fig. 1, page 540, both representing obstructive deafness, and see if there is much likeness. Again, compare the audiogram on page 534 with that on page 541, both representing perception deafness. In the former, the patient is represented as actually having better than normal hearing up to and somewhat beyond 1204 double vibrations, and on page 541, the hearing is represented as extending beyond the line which separates audition from feeling, for tones about and above 8192 double vibration.

Those of you who were present at the meeting of the Chicago Laryngological Society last Monday evening may recall the variability of the audiogram findings of Dr. Burch in cases of obstructive deafness. I trust he will find it convenient to enlarge on what he started to tell us the other evening, before his time expired.

On page 527 of the Audiometer number of the Laryngoscope, occurs the following paragraph "In the mixed type of cases there will be a decided drop at both ends." This finding corresponds to Goldstein's Audiogram of otosclerosis, which he presents on page 543 and repeated for us on Monday evening. Continuing the paragraph on page 527, it says "When there is a marked difference between the two ears of the same patient,

the sound may be perceived in the better ear before it is heard by the poor ear, then being tested."

I ask anyone here who has been in the habit of making careful functional hearing tests what this lateralization means. Lateralization to the better hearing ear, in the presence of nerve deafness, cannot be due to anything short of borrowed *cranial* conduction. A further proof that cranial conduction must play a role in testing the hearing power with the audiometer is the fact, as told us repeatedly by those with pronounced obstructive deafness, that they have little or no difficulty in hearing at the telephone. If these two observations are correct, it follows then, that when one takes the hearing of a patient with the audiometer, he is not testing the air conduction of a single ear, but the combined air and bone conduction of the side to which the receiver is put, plus the bone conductor of the opposite side; that gives us variable quantities, which in scientific research are disturbing factors, and which must be eliminated if accuracy in findings or conclusions are to be obtained.

Unfortunately, all the audiograms presented in the Larynscope are of bilateral conditions. It would be interesting to see a set of audiograms made of unilateral conditions. I feel that in such cases the influence of the borrowed bone conductor factor could be demonstrated to better advantage than in bilateral conditions of the same kind and nearly the same degree, as depicted in the audiograms already presented.

Again, when showing audiograms in the future, it would be well to parallel them with the tuning fork tests findings.

DR. H. V. DUTROW, Dayton, Ohio: I was surprised, when I received my program, to find that the program committee had put me down to discuss Dr. Fletcher's paper. I suppose the reason for their act was due to the fact, that I happen to be a member of the committee for the hard of hearing, and therefore should be expected to know something about deafness.

After listening to Dr. George W. Mackenzie's discussion, we are only once again reminded of the many criticisms ever besetting the road of scientific progress.

I wish to congratulate Dr. Fletcher upon his scientific accomplishment in perfecting a new apparatus for testing and charting, in percentages, the degree of normal hearing. It is of vital interest to every member of the Academy who numbers among his patients individuals with defective hearing. I shall not attempt to discuss the laws of physics and of higher mathematics which were necessary for him to correlate in the production of such a highly scientific apparatus as the audiometer. Dr. Fletcher is the pioneer in this work, and I am happy to "Render unto Caesar the things that are Caesar's," so I shall therefore confine my remarks, as far as possible, to the new line of thought suggested by his new apparatus in the field of otology.

In our quest for research workers in sound conducting apparatus, it was perfectly natural to look to the laboratories of the telephone and radio companies for aid along this line. This is only one example of countless instances in which the laboratory is essential to progress. It has recently been stated that no corporation or industry, not strictly financial in character, is larger than its laboratory. No corporation or industry today can meet competition without an adequate scientific research depart-

ment. Progressive medical men years ago realized that they could advance only by and with the aid of the laboratory.

Dr. Fletcher has been able to give us, through process of scientific deductions and mathematics, what he terms a sensation unit, and it is by this unit that he is able to calculate in percentages the loss of hearing in a given case. The terms threshold of feeling and threshold of hearing are most interesting. The points where the effect of the stimulation is just discernible are called the threshold of feeling and the threshold of hearing. The space between them represents the intensity range of hearing. That the threshold of feeling can be so extended as to produce acute pain, even though the patient may be entirely deaf, is most interesting and at once suggests the possibility of developing new mediums or transmission lines for sensation to the brain, by the intelligent and systematic stimulation of peripheral nerves. A practical demonstration of this possibility was revealed to us last evening by the results obtained in the training of the children presented by Dr. Goldstein. The essayist has given us an instrument whereby we can correctly obtain a uniform record, and at the same time make a differential diagnosis as to the various types of deafness. One is at once struck with the varied findings of our present methods of testing hearing of patients by the aid of tuning forks, watch tick, Galton whistle, voice and so forth; although after years of experience, we are very often able to achieve more or less uniform practical results, depending upon individual interpretation. These interpretations, however, would probably have as many variations as there are otologists making examinations. An instrument by which the vibrations are maintained uniformly at a given rate is certainly to be desired, instead of the tuning fork in which the rate of vibration is inconstant. Standardization of tests, and especially the records, is greatly to be desired. The audiometer, in addition to its accuracy, should also be a great time saving device, in that it is possible to examine a greater number of individuals singly, or in groups, in a shorter space of time. It should also be of value in medicolegal and industrial cases involving compensation for loss of hearing, and in detecting malingerers. With the advent of this new apparatus, it might be desirable to change the long used terms "air conduction" and "bone conduction" to "conductive audition" and "perceptive audition" respectively.

With all the mechanical advances, do we not still have the same dismal therapeutic problem before us? As otologists, we are interested in the treatment of the deafened. In order for scientific discoveries to aid us in the solution of the treatment of deafness, it is needless to say that they must be brought to the point where they can be put into practical application. I was intensely interested in the good news brought to us last evening by Dr. Norval Pierce, viz., that a committee of the American Otological Society had recently been successful in obtaining financial aid from the Carnegie Foundation, extending over a period of five years, to defray, in part at least, the expenses of an intensive research study of the disease known as otosclerosis. Prof. Nager, this morning, in his masterly presentation of his advanced study of deaf-mutism and his accomplishments, should be a strong incentive to our workers who are about to enter this field of scientific investigation. Would it be expecting too much, ere

another year or two passes by, to hope for progress in many forms of treatment of the deafened comparable to the work of Dr. Fletcher?

Dr. F. W. Kranz, Riverbank Laboratories, Geneva, Ill.: I think that, perhaps, the two points of particular interest in Dr. Fletcher's paper, so far as the physicist is concerned, are two of the definitions of units which Dr. Fletcher proposes for universal use. In the first place, he proposes what he calls a "sensation unit," this being a unit of attenuation or of difference in intensity between two sounds. I think that it is fair to say, and should be said, that this particular unit is a heritage from telephone practice. The telephone people were the first to become interested in attenuation, in how much speech was attenuated or reduced in intensity in going over a telephone line. If you talked from New York to Chicago, how much weaker would the speech be at the receiver in Chicago than at the transmitter in New York? To express the reduction in intensity, they used as a unit the attenuation due to one mile of standard cable—a telephone cable of a certain size and with certain electric constants. From measurements and calculations on this cable, it was determined that an attenuation expressed in miles of standard cable was 10.56 times the logarithm to the base 10 of the ratio of the initial to the final intensity.* That is:

$$N_{cm} = 10.56 \log_{10} \frac{J_1}{J_2}$$

where N_{cm} is the number of cable miles of attenuation and J_1 and J_2 are the initial and final intensities respectively. But for anyone not interested in the traditional background of this telephone unit, 10.56 would seem to be an odd constant to inject into the definition of a unit. About two years ago, the telephone people decided to drop the .56, making the unit that which is used here by Dr. Fletcher. The number of units now equals ten times the logarithm of the ratio of the intensities.

However, if the physicist would start out without any preconceived ideas to define a unit of attenuation, he would undoubtedly say that the number of units should equal the logarithm of the ratio of intensities. That is, he would leave out the 10 as well as the .56 from the expression given above. One unit would then mean an intensity ratio of 10, two units of 100, three of 1000, etc. There is a possibility, that, instead of logarithms to the base 10, which are the ones ordinarily used and which are used in the above discussion, a unit of attenuation could be based on the logarithmic base "e," thus using the socalled natural logarithms. In such a system, a unit of attenuation would correspond to an intensity ratio of 2.718. The use of such a unit was once attempted but sank from its own weight, as no one could be persuaded to accept a system based on such an unhandy number. No one is interested in this ratio, and it has no place in a definition of a practical attenuation unit.

Dr. Fletcher says that the unit which he proposes, ten times the logarithm of the intensity ratio, is such that one unit is about the change of intensity that a person can distinguish. This may be, but I do not see that this could have exerted any great influence on the choice of the unit. It is, perhaps, fortunate that an approximate correspondence in this

*See page 11 of "Transmission Circuits for Telephonic Communications," by K. S. Johnson.

regard exists, but it is not exact in any strict sense. So before such a unit is adopted universally, I think that it should be carefully considered from the standpoints of and by representatives of all those who may be expected to use it.

Another definition proposed by Dr. Fletcher, is that of the percentage of hearing of a person, a definition first given by Wegel, I believe. With a proper method of definition, if you should say that one man has 80% hearing and that another man also has 80% hearing, it should mean that the two have equally useful hearing. The percentage calculated on the basis described by Dr. Fletcher and his predecessors includes the consideration of frequencies up to 8000 cycles per second and above. But so far as useful hearing goes, a person may be stone deaf to frequencies above 3000 cycles or even above 2500, or even to all frequencies above 2000 cycles per second, and still he will be able to get along all right with conversation. If you should test such a person, however, by the use of separated speech syllables, you would find that there were some which he would not hear or understand; but in order for a person to understand a conversation, it is not necessary for him to get all of the sounds. However, as the percentage of hearing has been defined, these high frequencies have a weight comparable to the lower frequencies, although the latter are certainly much more essential for conversational purposes. In support of this viewpoint, it may be mentioned that the "loaded" telephone lines, which are arranged with Pupin coils, eliminate the frequencies above 2300 cycles. There is found to be no objection to this from a commercial standpoint. The carrier current telephone lines eliminate all frequencies above 2000 cycles per second, and yet they are perfectly satisfactory so far as transmission of speech is concerned. If percentages of hearing are to be used, they should be defined in such a way, that one person with 70% and another person with 70% would actually have equally good hearing. Certainly, a person with 70% should not have better useful hearing than a person with 80%, and yet with the method proposed, such a condition would be possible. A proper definition of percentage of hearing for speech would have to consider the practical matter of context and continuity in conversation, and such a percentage could not be determined with any great accuracy.

I hesitate to say anything about the otologic side of this paper, as you people know more about that than I do. Of course, the use of the audiometer by the practical otologist has to meet the question, "Does it help the otologist help the deaf to hear"? This question must be answered by the otologist, not the physicist. I might suggest, that an outline of the whole question might be made something in the following fashion: First, what are the methods of treatment of deafness which are available to the otologist? Next, what type of deafness is each applicable to? Next, how can the otologist most certainly and most easily distinguish between types of deafness for which different treatments are indicated? For diagnosis, the identification of the type of deafness is the information which is useful, and some otologists may feel that it is unnecessary to spend time and money in obtaining information of which they can make no use as far as betterment of the patient's hearing is concerned. For the purpose of tests on the progress of deafness, progress in either a positive or a negative sense, the audiometer is well suited.

From the research standpoint, I am perhaps prejudiced in favor of the sliding scale audiometer. It certainly gives information which cannot be obtained in any other way. In the audiometer charts which have been given by Dr. Fletcher, the points obtained at different frequencies are connected by dotted lines. This involves the totally unjustified assumption that something is known about the hearing in between the frequencies actually tested. By tests with the sliding scale audiometer, I have been able to show that there are often sharp breaks in the acuity of hearing as the frequency is gradually changed. There are deficiencies of various types, some covering only a narrow band of frequencies and some of considerable extent, some showing a gradual variation of acuity and some depressions with very steep sides, indicating abrupt changes in sensitivity of the ear mechanism. The presence of most of these would not be suspected if tests were made at only a relatively few separated frequencies.

As to the testing of bone conduction, it is quite possible to use an electric audiometer for this also. Dr. Fletcher did not show any means of testing bone conduction, but possibly he did not have a sufficiently powerful audiometer. We have made, at the Riverbank Laboratories, a considerable number of tests of bone conduction under various conditions with the electric audiometer, with interesting results, and such tests will undoubtedly be possible with the ultimate audiometer.

Dr. Austin A. Hayden, Chicago: I have been using an audiometer for the last six or eight weeks, and in that time have examined about 100 ears, that is to say, about 50 patients. One was a case of a man recovering from an acute otitis media, which he thought was not clearing up as fast as it should. He complained of a considerable amount of deafness. Aside from making the ordinary fundus examinations, I put him through the audiometer tests, and discovered that his loss of hearing was confined almost entirely to the upper scale. We, consequently, suspected that his deafness was not due to the acute otitis media, but to some nerve lesion. Whether the nerve lesion followed his head cold and otitis, or whether it was preexistent, was of course something we could not determine; but we were able to give a very accurate prognosis in regard to his hearing, which was justified by results, because his hearing has not improved since. I mention this, because I think many more routine hearing examinations will be made in cases of that sort with the audiometer, and a more definite prognosis can be made in a way not possible with tuning forks, because an instrument of this sort can be used by many assistants and quite accurate routine examinations made.

In regard to bone conduction, Dr. Fletcher did not show the apparatus than can be used with a small office audiometer for bone conduction. I hope he will speak of that in closing.

Another thing of practical importance that has occurred to me, is that it would be very much better to make the headpiece to fit over the head like that of a telephone operator, rather than over only one ear. If that were done, the sound would be thrown into one or the other ear with a switch which would be an invaluable aid in testing malingering.

Another thing that I am sure has occurred to almost everyone here, is the limitation in range of the middle size audiometer. As you know, it is confined to vibrations from 64 to 8192, not covering as wide a range as

many of you would like. It seems to me, it would be a distinct advantage to have the machine cover at least down to 32 and, perhaps, up to 16,000 double vibrations per second.

I must say that my experience with the machine has been very, very satisfactory, and I look for a very wide use of it among the members of this Academy, and a very gratifying use. I think the Western Electric are to be congratulated on the work they have done.

DR. HARVEY FLETCHER, New York City, (closing): I think the remark is a propos, that progress with the audiometer is made with considerable hazard. It is fine to get criticism. Probably it is a good thing to get destructive criticism, but it is much easier, let me tell you, to criticize a thing than to get busy and make something that is better.

I will try to answer some of the questions which have been asked. There were a number of complimentary things said, and a number of things said that seemed to be obvious, which of course will need no comment from me. Dr. Mackenzie wondered why a person with a considerable degree of deafness could hear over the telephone. In using the telephone, you may get sounds from the receiver about a thousand times more powerful than those which are required to give good audition under conditions of no noise. What does this do for the deafened person? The deafened person, who has perhaps 30 per cent loss of hearing, can hear over the telephone under these conditions because the speech is delivered to him with 30 units more loudness than is required by a person of normal hearing. The reason he can hear is because the speech is louder.

Regarding the relative amounts of air and bone conduction produced when a telephone receiver is held to the ear, the question raised here today has been brought up by nearly every doctor who has come in contact with the audiometer. The first thing he asks when he puts the receiver to his ear, is "Will it cause bone conduction?" You are receiving sounds by bone conduction now as the air waves strike against your head. The question is, how much is the bone conduction compared to the air conduction? When you hold the prongs of a tuning fork to your ear to make the usual air conduction test, a small part of the sound is transmitted to the cochlea by bone conduction. The same thing is true of the telephone receiver—a small part of the sound is conducted by bone but the far greater part is conducted by air. Before recommending such an instrument for use, we had to make sure that the bone conduction was a very small part of the air conduction. When you hold the receiver to the right ear, it takes about a million times as much intensity to make the left ear hear as when it is held directly to the left ear. That is, only one millionth as much energy goes to the cochlea of the opposite ear as reaches the cochlea of the ear upon which the receiver is placed. Now you are all familiar with the fact, that when the handle of a tuning fork is held on the mastoid bone, the sound is heard about as well in one ear as the other. This shows that the bone conduction from the telephone receiver is a very small fraction of the air conduction.

The two receivers suggested by Dr. Hayden are now available. You can switch from one to the other by means of a switch in the cord. Also you can put these small rubber ear pieces over the caps of the receivers if you want to. They are a help in excluding outside noises.

Dr. Kranz referred to the fact that these sensation units are the same that the telephone people are using in transmission engineering, as though it was wrong to borrow something found useful in telephone engineering and use it for describing the degree of deafness. Since he has revealed this secret, let me reveal another. Dr. Kranz received his splendid training while in the Bell Telephone Research Laboratories, where he worked for a number of years. Because these units are used in telephone engineering, is one of the reasons why it is desirable to use the same units in measuing a similar thing in otology; that is the reason why we have discussed it with psychologists in an endeavor to get them to use the same unit. It is because this unit has proved satisfactory in measuring the loudness of received speech, that it was suggested to the otologic profession as a convenient unit. It is convenient and practical, because it is the right size for measuring noticeable changes in hearing, and equal changes in hearing loss on this new scale correspond to equal changes in the loudness of sound that is required to make the deafened hear. No matter what I say, or what Dr. Kranz says, or what the committee on standards says, the thing that will ultimately be used is the thing that you will find by actual use is the most practical and serviceable.

The per cent of hearing scale, Dr. Kranz thinks, is of no value because it is not definite. I tried to make it evident in my paper that the per cent hearing does not mean anything if you say per cent of hearing and nothing more. If you say per cent of hearing for the tone of pitch 256, then every patient who has 30 per cent of hearing for 256 would have the same hearing ability. For scientific purposes, I stated that it is better not to use per cent hearing, but to express the hearing loss in sensation units. But you are compelled to use something to satisfy patients who will not understand the term sensation unit, and under such circumstances the best thing is per cent hearing loss. The patient wants to know the per cent hearing loss for speech which may be calculated from the audiogram or obtained directly by speech tests.

In regard to the number of observed points which are necessary to determine an audiogram, Dr. Kranz suggested that the audiometer for research purposes should have a continuous pitch change. Whether this is true or not depends upon how many points you are going to use for plotting the audiogram, and the answer to this question will be obtained from practical experience. For some research purposes, it is probably advisable to have the points closer together than would be obtained directly with the audiometer. This is made possible by the use of an auxiliary condenser. Ideally, the points should be so close together that the ear just perceives there has been a change of loudness or pitch in going from the condition represented by one point to that represented by another. How near this ideal one desires to go depends upon the purpose one has in view.

In regard to the range of frequency, if we could have an instrument that we could carry with us in our vest pocket, and which would give the double or complete vibrations from 16 to 16,000, it would be fine; but this is impossible. So we will have to be content with the best compromise for practical use. In my judgment, the use of the 2-A audiometer will indicate to the otologist that it is not necessary for diagnosis to make tests either at the very low tones or the very high tones. By getting most

of the intermediate range, you can tell pretty well what will happen at the high and low ranges. It has been my experience, that those who use the audiometer put less and less stress on the high and low frequencies. In the middle ranges, you can make the measurements more accurately.

Now, I want to say something about the bone conduction receiver. Unfortunately, I forgot to bring one with me. It can be used on the audiometer by substituting it in place of the usual air conduction receiver. With any bone conduction receiver that is now available, it is difficult to make accurate measurements above pitches corresponding to 1000 vibrations per second for the same reason as it is difficult with the tuning forks, but in the middle ranges you can make an audiogram by bone conduction as well as air conduction. When these are correlated with your other clinical information, you will realize the significance of these audiograms.

WHICH SEPTA DEMAND OPERATION?

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MINNEAPOLIS, MINN.

The technic of a septum operation is now practically standard, but which septum to operate is in dispute.

In prehistoric times, the principal function of the nose was undoubtedly olfactory, as this sense was necessary for protection of the individual; but through evolution, changes have gone on until now the principle function is respiratory.

In our modern way of living in overheated houses, and with varying temperatures out of doors, this new principal function of modifying the air on its passage through the nose and filtering out dirt and germs, has become intensified and important.

The ethmoidal sinuses formerly contained turbinates covered with olfactory nerves. They are now only rudiments of these important structures, which drain and receive aeration, together with the frontal and maxillary sinuses, through the nasal cavity proper.

The nasal chambers are also really continuations of the Eustachian tubes, so that the ventilation of the middle ear is practically through the nose.

The work of modifying the air is performed principally by the membrane covering the turbinates; therefore it is very essential that these structures be left in place. The septum is merely a fixed wall towards which the membrane covering the turbinal bones swells or contracts to enlarge or narrow the nasal space as required.

The ideal nose, then, has a more or less thin, straight septum, the nasal cavities are equal in size and the turbinates are in place, so that both sides are doing an equal amount of work in modifying the air on its passage through the nose.

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Nasal septa are classified under three headings: normal, deflected and thickened.

Deflected septa have no definite classification because of their variety. The deflection may be in the cartilage or bone or both, up or down, forward or back, but as a rule, more or less follows the sutures. The socalled spurs are part of a deflection.

The thickened septum receives most consideration when the thickening is between the middle turbinates.

Deflected and thickened septa are the primary causes of changes within the nasal cavities.

Following a deflection in the septum, Nature, in trying to correct the spacing of the nose, thickens the membrane of the turbinates on the concave side and thins the membrane on the convex side, thus forming a compensatory hypertrophy of the membrane of the turbinates on the concave side, and a compensatory atrophy on the convex side. On neither side is the membrane of the turbinates normal.

Under these conditions, the air, on its way through the nose, is not properly modified, which leads to constitutional changes.

Patients become undernourished, thin, nervous and lacking in constitutional vigor.

A deflected septum, or a septum thickened high up between the middle turbinates, besides causing changes in the membrane covering the turbinates, crowds the turbinates against the lateral wall and interfere with the drainage and aeration of the sinuses, subjecting the patient to possible sinusitis.

These changes within the nose cause a more or less congested condition, making the membrane easily susceptible to infections, especially "colds."

If drainage interference continues long enough or is repeated often enough with the right infection, all the changes of sinusitis follow, from the acute to the chronic stages, with pus, polypi and attending symptoms.

A congested nose, either continuous or intermittent, causes a congestion of the membrane lining the Eustachian tubes, and leads to pathologic changes within the middle ear.

Assuming that an ideal nose is essential for free breathing, drainage and aeration, this alone is not a sufficient reason for

operating a septum which does not come up to this standard.

It is true that most people have a more or less deflected or thickened septum, with apparently no symptoms so far as they can judge.

No one can tell positively how much effect a not normal nose has on general constitutional conditions, but certainly the nose should be taken into consideration in all cases of so-called nervousness, and in thin, undernourished patients.

I believe that nearly every case of sinusitis is due primarily to a deflected or thickened septum, which interferes with drainage and aeration.

Certainly every case of repeated acute attacks of sinusitis, that is, a congestive sinusitis with pain, should have the septum carefully inspected as to a deflection or thickening in the region of the middle turbinates. Practically all chronic sinusitis cases with pus and polypi need a submucous resection together with the sinus operations, if we expect to benefit these patients permanently.

These seem radical statements to make, but I believe one of the failures in the cure of sinus cases is due to the lack of this operation. Not properly studying the cases is the only reason I can give for not observing these points.

In cases of middle ear pathology, the nose must be free if we expect proper aeration through the Eustachian tubes.

A complete submucous resection must be performed in every case, and particularly must the bone be well taken out between the middle turbinates.

It is surprising what poor submucous resections are performed even by our socalled good men, and it is no wonder they get poor results. This is probably not due to their lack of surgical ability, but rather to their surgical judgment.

My contention is that in too many of these cases the bone is not fully removed in the region of the middle turbinates, and too much bone is left above or below, causing tenting of the membrane.

Instead of too many submucous operations, not enough are being performed, if we study these cases properly and expect to get results.

But above all things perform a complete operation.

Summary.

The following class of cases should be considered for septum operations, to obtain proper modification of the air passing through the nose, and for drainage and aeration of the sinuses and Eustachian tubes:

Socalled nervousness;
Thin and undernourished patients;
Mouth breathers;
Patients who have repeated colds, or "stuffy" noses;
Repeated acute congestive sinusitis;
Chronic sinusitis with pus and polypi;
Asthma;
Hay fever;
Middle ear pathology.

DISCUSSION.

Dr. J. J. Shea, Memphis, Tenn.: I wish to congratulate Dr. Pratt upon his excellent paper, which deals with a subject that is of greater importance to the average member than a thesis upon a rare surgical or pathologic condition. The submucous resection of the nasal septum is fundamentally a plastic operation and not a radical procedure, inasmuch as the parts subjected to the surgical procedures should retain their normal physiologic function. By the foregoing statement, I mean that the septal membrane should still be capable of modifying the inspired air to meet the physiologic demands when the parts have healed. If the resection is carried up as high as the superior turbinate, the olfactory sense will be lessened. At no time should the resection be carried into the olfactory mucosa, if the superior turbinates are to be removed, as this will destroy the sense of smell. The most favorable results are obtained when a case of the border line variety is operated upon. In fact, the damage done by extensive deformities lessens the possibility of much improvement, whereas the septum that is thickened high up, but not deflected in its lower half, does not produce permanent obstruction to the ventilation and drainage of the ethmoidal cells, but does produce an intermittent obstruction whenever the septal membrane is congested. After the resection of the bony and cartilaginous septum, this congestion of the membrane is not so capable of blocking the sinuses. Of late, it has become popular not to resect the septum but to exenterate the ethmoidal cells. Many of the sinuologists will find that a resection of the septum before their sinus operation may delay or prevent the operation, and if the sinus operation is necessary, a straight septum will afford them a better view for the careful removal of the ethmoidal cells and a freer drainage during the postoperative period. There are septums, around which the ethmoidal cells can be exenterated that should be resected to prevent future blocking during subsequent colds, and to afford sufficient space for postoperative treatment of the ethmosphenoidal labyrinth. When the nose is thoroughly shrunken in preparation for an ethmoidal operation, the septum may appear to be innocent,

but if this septum, during its normal state of vascularity, offers an obstruction to thorough drainage and ventilation, it should be resected. In fact, I feel that we do not resect sufficient of the septums that come to us for treatment, for we are inclined to pass up the border line cases, whereas the resection of this septum will give the patient proportionately a greater benefit than a resection of a badly twisted and deformed orthopedic septum.

Dr. Edwin McGinnis, Chicago: I feel very well qualified to discuss operations on the nasal septum, because I have had four submucous resections done by three different men. The first was the earliest type of operation done. I remember how nicely the operator did it. I had no hemorrhage, there was no pain, my nose was stuffed full of gauze, which was taken out the next day, and I thought I would be all right. But in three or four weeks, it was the same old story. I had to go through two more operations on the nasal septum, which gave some relief, but the one sided headaches still continued. The last attempt cleared up the headaches, and I have a free nose at the present time.

In discussing this subject with a well known general surgeon, he said there are too many septum operations being done; that if the septum is bent over to one side, the other side will take care of the need for extra air. He did not know anything about compensatory hypertrophy.

I had two patients in the office a couple of weeks ago. One had a submucous done that was a perfectly good job. The septum was straight, both above and below, but yet the patient said she could not breathe through her nose at night, and that the headaches for which she had sought relief were still present. She had a purulent ethmoiditis, and the secretions were obstructing her nose and causing pain. So remember that a septum operation must be followed up to make a satisfied patient.

Some of you know that I never take out any turbinate tissue. I like to save all of the nasal mucous membrane. The only way to do that in certain types of noses is to get the septum away from the lateral wall. In the last two weeks, I have seen two patients who had complete removal of the ethmoid cells and the middle turbinates, and the only thing left in the ethmoid region was the lacrimal plate and the roof of the ethmoid. These operations were done around a markedly curved septum. I congratulate the surgeon, because he took a lot of care to go around the septum and get the ethmoid tissue out. I think it is better to do the septum operation to get space to drain the ethmoid cells underneath the turbinate. With this procedure, there is not so much disturbance of physiologic function.

Dr. Cullen F. Welty, San Francisco: I have been talking along this line for several years past, and I wish only to accentuate what the speakers have said—that we do not go high enough in our septum operations. When is it necessary to go high? How do you determine that factor? This is my indication for such an operation. After the parts have been cocainized and I see a contact—that means something to me, either in a hay fever case, a neuralgia case, an asthma case, headaches, or simply a dropping in the postnasal space.

I wish to say, furthermore, that the middle turbinate must hang free. If you wish to do an ethmoid operation, whether you leave the middle

turbinate or take it away, you must first correct the septum. It is bad surgery to do it the other way.

I have only nice things to say about the complete removal of contacts. Get rid of your contacts and your end results will be more satisfactory. I am very enthusiastic about this particular procedure.

Dr. Frank R. Spencer, Boulder, Colo.: Several types of septa demand operation: 1. I hardly need to mention the nasal obstruction produced by marked deflection. 2. Pressure against the outer wall of the nose, or pressure against a turbinate will produce headache and hyperesthetic rhinitis. 3. One type that has not been sufficiently emphasized is the one associated with chronic suppurative otitis media. 4. A septum that interferes with drainage of the sinuses demands operation. 5. Another factor that has not been given sufficient importance, but which Doctor Mosher has repeatedly emphasized, is the importance of straightening the septum prior to an operation on the tear sac. Some of the failures have been due to negligence in not operating on the septum at the same time the anterior ethmoidal cells are removed.

I believe that sometimes too many septum operations are done, but on the other hand we see patients who have gone a long time with deflected septa that need to be straightened.

Dr. B. N. Colver, Battle Creek, Mich.: There is one point touched upon by Dr. Welty, that I wish to emphasize and that is the correction of the turbinates in their relation to the new plane of the septum. If one looks at the turbinate on the convex side of a markedly deflected septum, it is found to be atrophic, thin, and bent in to fit the curve of the lateral wall of the nose. If it is infracted away from its bed and straightened with compression forceps, you will be surprised in a year to see how it has actually become thicker. On the other side, the concave side of the septum, very often the turbinate is hypertrophied, but more often it is cellular (so called cystic). In these cases, the straightening of the turbinate after the removel of the lateral half of the cell will reduce it to one-third or one-fourth of its lateral diameter. When this turbinate is given a position parallel to and equidistant from the lateral wall and the new septal plane, you will be surprised in a few weeks to see it look as if it had always been a normal turbinate.

Dr. Fred J. Pratt, Minneapolis, closing: The thing I want to emphasize is that the septum is the primary cause of most of the troubles in the nose. It is rarely necessary to do anything to the turbinates after a septum operation, because the atrophied turbinate will enlarge and the lypertrophied turbinate will shrink if you give them time. The only turbinate that needs attention would be the cystic.

Also I wish to repeat that not enough septum operations are being performed—but we must do good ones. Incomplete resections do not give results, and these patients go from one doctor to another, complaining about their operations. When doing a submucous resection, do a complete operation.

RECENT DEVELOPMENTS IN PERORAL ENDOSCOPY. ESOPHAGOSCOPY AND BRONCHOSCOPY FOR DISEASE. REPORTS OF CASES.*

GABRIEL TUCKER, M.D.†

PHILADELPHIA.

Bronschoscopy and esophagoscopy originally concerned, chiefly, in the removal of foreign bodies, have developed great usefulness in the diagnosis and treatment of disease, by bringing under direct inspection of the eye regions of the air and food passages formerly invisible. The following reports of cases illustrate phases of the development of peroral endoscopy.

POSTOPERATIVE MASSIVE COLLAPSE OF THE LUNG.

In recording the observations from the bronchoscopic examination of two cases of postoperative massive collapse of the lung, nothing new is offered as to etiology. The literature has been recently reviewed and the subject very ably considered by a number of observers, among them Dr. Walter Estell Lee[1], of the Pennsylvania Hospital, Philadelphia; Dr. J. W. Scott[2], of The Peter Brent Brigham Hospital, of Boston; Dr. Edward D. Churchill[3], of The Massachusettes General Hospital, Boston; and Dr. Simon Leopold[4], of the University of Pennsylvania Hospital, Philadelphia. Of the two cases here reported, the first occurred in a boy, aged 13 years, on the service of Dr. Walter Estell Lee at the Germantown Hospital, Philadelphia. This is believed to be the first case of postoperative massive collapse to be examined bronchoscopically. The examination was made January 14, 1925. That bronchoscopic study might be able to throw considerable light on the mechanism of postoperative pulmonary collapse, had

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been suggested by Chevalier Jackson to Dr. Simon Leopold, who recorded this suggestion in his paper on the subject of Postoperative Massive Collapse, but opportunity for bronchoscopic study had not presented itself until, at Dr. Lee's request, a bronchoscopy was done for this condition. A boy, thirteen years of age, had been in good health until he developed acute appendicitis with abscess. He was admitted to Dr. Lee's service at the Germantown Hospital. The abscess was drained under ether anesthesia, the procedure requiring only a few minutes. Thirty-six hours following the operation, he became acutely ill with symptoms of shock. Physical examination showed the signs of collapse of the right lung. X-ray examination confirmed these findings. There was the typical displacement of the heart toward the affected side, with evidence of increase of density of the affected right lung. The patient reacted from the acute symptoms, and bronchoscopy was done 48 hours after the onset of the collapse. There had been expectoration of the typical tenacious sputum, slightly greenish-gray in color, and when expectorated into a cup, it stood up from the bottom of the cup like gumdrops. On bronchoscopy, the tracheal mucosa was found reddened and glairy, greyish secretion adhered to the tracheal wall. The inflammatory condition became more marked in the lower trachea and right bronchus. The left bronchus was clear of secretion, the mucosa being only slightly inflammatory. The orifice of the right main bronchus was completely surrounded by a thick ring of tenacious secretion. The orifice of the right upper lobe bronchus was not completely blocked, but patches of secretion were adherent to the wall of the bronchus. The stem bronchus and the middle and lower lobe bronchi were completely blocked with the thick secretion. The mucosa was very red and thickened. The secretion was aspirated from the larger bronchi.

It was noted that the right main bronchus deviated toward the right; there was no evidence of bronchial compression. There seemed to be marked restriction of the bronchial movements. Inspiratory opening and lengthening of the bronchus was very slight, particularly in the lower and middle lobe bronchi. Bronchoscopic diagnosis was diffuse bronchitis involving the right main bronchus and its branches, most marked in the lower stem and middle lobe bronchi; bronchial obstruction of the middle and lower lobes with thick tenacious

secretion. Swab specimen of secretion showed "pure culture of pneumococci."

Physical examination immediately following bronchoscopy showed evidence of air entering the right lung. The heart showed less displacement to the right. Within 24 hours, the collapse had recurred. At the end of 48 hours, the right lung had begun to clear; the character of the sputum had changed, being mucopurulent, thin, and less tenacious. Bronchoscopy was repeated at the end of the third day and showed the inflammatory condition of the trachea and right bronchus to be much less severe than at the previous examination. A small amount of secretion, slightly purulent in character, was aspirated from the right main bronchus; the left bronchus was free of secretion. The right bronchus showed no abnormal deviation, and the bronchial movements seemed normal. The secretion in the middle and lower lobe bronchi was thin and was aspirated without difficulty. The bronchoscopic findings indicated that there was a much larger degree of aeration of the lower and middle lobes than at the first bronchoscopy. Previous to this bronchoscopic examination, the heart had gone back to practically the normal position, and the physical signs indicated that there was good aeration of the right lung. The recovery of the patient was uneventful following the second bronchoscopy. It was noted by Dr. Lee that the collapsed lung cleared within 72 hours after the first bronchoscopy, while in the other cases of his series, in which bronchoscopic treatment had not been carried out, the recovery had extended over an average period of twenty-one days.

Case 2: The second case occurred on the service of The Chevalier Jackson Clinic at the Jefferson Hospital. A boy, 6 years of age, had been operated on for the closure of a gastrostomic fistula that had been made for the purpose of retrograde treatment of cicatricial stricture of the esophagus. The stricture of the esophagus was cured and the gastrostomy opening had been closed. Ether anesthesia was used during the operation, the patient having a great deal of cough and considerable secretion during the anesthesia. Thirty-six hours following the operation, pulmonary symptoms developed; and by the end of 48 hours following the operation, the typical signs of postoperative massive collapse of the left lung were present. Seventy-two hours following the operation, bronchoscopy was done, June 13, 1925. Very thick, tenacious

secretion, whitish-yellow in color, was found in the trachea and left bronchus. The mucosa of the bronchus was very inflammatory. Following aspiration, air seemed to enter the left lung. Ten c. c. of the thick, whitish-yellow exudate were aspirated, and a culture of the removed secretion showed pneumococci and staphylococci. Dr. Manges' report of roentgenogram on June 13, 1925, reads: "There is complete collapse of the left lung; trachea and heart are drawn well to the left. It is impossible to see the shadow or outline of the heart or left diaphragm. The lung shadow is equal in density to that of the heart so that there is no detail at all seen in the left chest." Films made one and a half hours after bronchoscopy show: "The lungs contain a considerable quantity of air. The trachea is near the median line, the right border of the heart is even with the right border of the spine shadow, and the outline of the diaphragm as well as the left border of the heart are clearly seen. There is evidence of function, because there is more air in the left lung at inspiration than there is at expiration. This indicates definitely that some obstruction was removed from the left lung at the time of bronchoscopy." The lung showed evidence of collapse again in 42 hours after the first bronchoscopy, and the X-ray examination gave the same findings as before bronchoscopy. Bronchoscopy was again performed, and 14 c. c. of thick, yellowish secretion were aspirated. X-ray examination again showed return of function in the lung. Three days later, the collapse had recurred and another bronchoscopy was performed. The secretion was less in amount and less tenacious in character. The aeration of the left lung remained improved for a considerably longer period, but there was still evidence of considerable secretion in the chest, and one week later bronchoscopy was again performed and four c. c. of the thick, tenacious material were aspirated from the left lung, principally from the lower lobe bronchus. Two subsequent bronchoscopies were done after intervals of two and five days respectively, and following this the patient was able to keep the lung free of secretion. The cough reflex seemed to have returned, his improvement was progressive, and he was discharged from the hospital well, six weeks following the onset of the postoperative massive collapse. In all, six bronchoscopies were done during a period of twenty days.

In the second case, the secretion was more purulent in character than in case one, which can probably be accounted for by the presence of the staphlylococci. The patient also seemed more toxic, and was very much less able to cough up the thick secretion.

We have demonstrated bronchoscopically that bronchial obstruction exists. The tenacious mucus, which Pasteur and practically all other observers state is coughed up in all cases, acts as a foreign body. Whether this tenacious secretion is produced by the collapse of the lung squeezing the secretion into the bronchi, or whether its presence there causes collapse of the lung due to the absorption of the air in the portion of the lung obstructed, does not influence the fact that it is necessary to remove the obstruction by aspiration, by absorption, or by liquefaction and coughing it up. In some manner it must be removed, so that air can enter before the lung can possibly expand.

Dr. Manges has proved by Roentgen ray examination that function is restored following bronchoscopic removal of the obstructing secretion, and we have been able to demonstrate by repeated bronchoscopies in the second case that the tenacious secretion blocks the bronchus when collapse is present. Bronchoscopy, after the collapse has disappeared, shows purulent secretion present but not obstructive in character or quantity.

We have seen massive obstructive atelectasis in many cases when the bronchus was blocked by foreign body. The foreign body that is the most effective in producing atelectasis is the dried bean. This object, owing to its shape when lodged in the bronchus, blocks the passage of air; and the action of warmth and moisture in the bronchus produces a rapid increase in the size of the bean, corking the bronchus completely. Atelectasis is very quickly and effectively produced. The physical signs and X-ray findings are the same as in post-operative massive collapse. The removal of the bean uncorks the lung and it rapidly expands, and in a few hours returns to normal. The obstruction is completely removed. In the cases of massive collapse where the secretion has been removed bronchoscopically, partial function is restored immediately, but it is not possible to aspirate all of the secretion from the finer bronchioles. The secretion reaccumulates in the larger bronchi, and the collapse recurs, due to the com-

plete plugging of the bronchi. The indication is clear for early bronchoscopic aspiration of secretion. The procedure is carried out with local anesthesia in adults and no anesthesia in children. The question as to what patients should be aspirated is one for the surgeon who has knowledge of the operative condition to decide.

When reaccumulation occurs, repeated aspiration should be carried out until the weakened respiratory force revives, the cough reflex returns, and the lung is able to rid itself of the obstruction; or in other words, until the vicious circle of obstruction atelectasis, atelectasis obstruction is eliminated.

Posttonsillectomy and Postoperative Pulmonary Abscess.

Posttonsillectomy and postoperative pulmonary abscess are now recognized as being a much more frequent occurrence than was formerly believed. The supposed "catching cold" following tonsillectomy or other operative procedures is now recognized as a direct complication following the operation. As to the mode of infection, some cases are undoubtedly due to aspiration of infective material at the time of operation. This is proven by the aspiration of deciduous teeth which have been dislodged at the time of the tonsil operation and inspired into the lung, with resultant abscess. Recently, the author removed bronchoscopically from the left bronchus of a patient a tonsil sponge that had been aspirated during the course of a local tonsillectomy.

If foreign material of this type can be aspirated, it is reasonable to conclude that infective material can be aspirated in the same manner, which would result in abscess formation. The infection may also occur through the blood stream, as has been proven by the work of Fetterolf and Fox and other observers. It would seem that bronchoscopic aspiration would be most effective in the cases in which the abscess is due to inspiration of infective material. We have not, however, been able to prove this in the bronchoscopic treatment of posttonsillectomy pulmonary abscess. The results of bronchoscopic treatment in many cases have been very encouraging; in a few cases, bronchoscopic aspiration has resulted in prompt cure. Our experience has led us to believe that the sooner the bronchoscopy is done, particularly in cases due to aspirated infection, the more promptly the beneficial results are obtained. Three cases are reported here in which

bronchoscopic treatment undoubtedly contributed very greatly to the cure.

CASE 1: Male, aged 40, tonsillectomy five months before admission to the clinic. Pulmonary symptoms commenced four days following tonsillectomy under ether anesthesia. At the end of the first week, there were elevation of temperature and cough with expectoration of foul sputum; and it was thought that he had "caught cold" following the tonsillectomy. He did not improve under medical treatment and, at the end of four months, he consulted Dr. Robert D. Spencer of Ashland, Pa., who, after X-ray studies and bronchoscopic examination, diagnosed the condition "pulmonary abscess involving the lower lobe of the left lung." When the patient came to the clinic, it was found that there was a pyramidal area in the left lower lobe that extended outward from the hylus of the lung toward the pleura, with the base outward, and physical examination showed there was pleural involvement. The surgical consultant advised observation of the patient, and if the condition should not improve, he felt that external drainage would be the proper procedure. At the end of a week, the pleural signs had cleared to some extent. X-ray examination showed there was a decided decrease in the area of pathology in the left lower lobe; particularly there had been a clearing up of the peripheral area of the lower lobe. The patient was kept under observation, and improvement continued. At the end of four weeks, he developed a fever, 101° F., and complained of sore throat. Examination at that time showed that he had an acute infection of the upper respiratory tract. Following this, the amount of expectoration increased, but the area of involvement in the lung, while larger, seemed to have cleared up at the periphery, at the point of pleural involvement. In consultation, it was decided that bronchoscopic drainage should be done. This was carried out at semiweekly intervals, and after eight bronchoscopies no sputum was being expectorated, the man had gained 25 pounds in weight, and repeated Roentgen ray examinations showed a progressive decrease in the area of lung involved. At the end of two months following the beginning of bronchoscopic treatment, the lung had cleared up completely. There is now no expectoration, and the patient has been discharged well. In this patient, there was a contraindication to bronschoscopy when he was admitted, owing to the pleural in-

volvement; and it was felt advisable to consider external drainage. On the improvement of the pleural complication, the contraindication was removed, and when the patient ceased to improve, bronchoscopic treatment was instituted and rapid cure resulted.

CASE 2: A boy, seven and one-half years of age, was referred to the Clinic seven weeks following tonsillectomy. The operation had been performed under ether anesthesia. In the first week following the operation, the child developed a cough, and had pulmonary symptoms up to the time of admission. He had lost weight, and there had been an afternoon rise of temperature to 101.4° F. A Roentgen ray examination by Dr. W. F. Manges, three days prior to his admission, showed an abscess in the lower portion of the right upper lobe. X-ray examination the day following the child's admission showed an increase in the area of involvement. Physical examination also indicated that there was an extension of the pathologic process. Diagnostic bronchoscopy was done, and evidence of a suppurative process was found in the right upper lobe. A swab culture taken showed a practically pure culture of pneumococci. There had been no expectoration prior to bronchoscopy, but following bronchoscopy there was considerable cough and expectoration of very foul pus. The child's general condition showed improvement immediately. Two days later, a second bronchoscopy was done and considerable quantity of very foul pus was aspirated from the right main and upper lobe bronchi. The pus was found to be coming entirely from the right upper lobe bronchus. Roentgen ray examination following bronchoscopy showed marked improvement. The abscess had been evacuated. The area of involvement in the right upper lobe showed a cavity one inch in diameter that was free of fluid. The patient's temperature came down to normal and remained there. Roentgen ray examination four days later was reported by Dr. Manges as showing a distinct diminution in the area of pathology. The improvement was progressive. At the end of a week, another X-ray examination was made, and Dr. Manges reported definite improvement, and that the inflammatory area was clearing up. The patient was discharged from the hospital, and his improvement continued to complete recovery.

CASE 3: Male, aged 30 years, a former enlisted man in the British Navy, was injured by the blowing off of the top of a pneumatic paint spraying machine that he was using, the top striking him on the neck, jaw and right chest. He had a fracture of the lower jaw in two places, and a number of teeth were dislodged. He was operated upon five weeks after the injury, under local anesthesia. A portion of the lower jaw was removed and several teeth were extracted. Four weeks later, a second operation was done and additional teeth were removed, with a second piece of the lower jaw, the procedure being done under gas and ether anesthesia. Two days later, he developed a severe pain in his chest, and five days following the operation he began to expectorate blood stained pus. The productive cough and septic temperature range continued during the next three weeks. X-ray examination prior to admission showed a pulmonary abscess in the right lower and middle lobes. He was referred to the Clinic by Dr. John S. Eynon, of Chester, Pa., for bronchoscopic treatment. Roentgen ray examination revealed no evidence of foreign body. Bronchoscopic examination showed very foul, blood stained pus in the bronchus and trachea. After aspiration, it was found to reappear from both the lower and middle lobes. The mucosa of the right lower and middle lobe bronchi was very inflammatory. Pus was aspirated and local medication applied; no foreign body was seen on bronchoscopic examination. The surgical consultant advised that bronchoscopic aspiration be carried out. The patient's blood Wassermann was negative. The culture from the swab specimen taken at the first bronchoscopy showed streptococcus mitis (nonhem.) to be the predominating organism. Vaccine was made from this culture and administered by the medical consultant. Bronchoscopy was repeated at weekly intervals, pus aspirated and local medication applied. The patient's condition improved progressively, and after five bronchoscopies the inflammatory condition of the middle and lower lobe bronchi had almost entirely disappeared. The pus had lost its odor and had become thin in consistency and whitish in color. The man's general condition had improved markedly, and he insisted on getting up around the ward, his temperature having been normal for a period of two weeks. He was allowed to go home and return as an outpatient for treatment. X-ray examination at this time showed marked diminution of area

of involvement in the right lung. Bronchoscopies were carried out at weekly intervals, and at the seventh, the third after he left the hospital, the bronchus was found practically normal and the tracheobronchial tree free of pus. The man had gained twenty-five pounds in weight, and X-ray examination at this time showed progressive improvement. Bronchoscopy was discontinued two months later, and the patient returned at the end of another two months for observation. He was found to be in excellent physical condition. His weight on admission had been 105 pounds, and at the time of his return it had increased to 152 pounds. X-ray examination showed the right chest practically normal.

The patient presented an unusual complication because of the fracture of the lower jaw. It was found possible to expose the larynx by working from the left side of the man's mouth instead of the right, the usual route, without discomfort or interference with the injured lower jaw; and in this manner the treatments were carried out. This demonstrates that branchoscopy can be done, as Jackson states, in any patient whose mouth can be opened widely enough to admit a bronchoscope.

Bronchoscopic Aid to Tracheotomy.

There are certain pathologic conditions, in which there is tracheal compression and displacement of the trachea, where bronchoscopic aid in maintaining the airway and finding the trachea for insertion of the tracheotomic cannula is invaluable.

Case 1: A man, aged 65, was seen in consultation with Dr. Frank Bridgett. He was extremely dyspneic and had a massive enlargement of the thyroid gland, malignant in character, which covered the larynx and extended downward to the upper thoracic aperture, bulging outward above the suprasternal notch. Mirror examination showed a bilateral recurrent paralysis, although the dyspnea was both inspiratory and expiratory. The question of tracheotomy had been considered, and it was thought impracticable because of the enormous size of the thyroid and the resultant tracheal compression and displacement. It was felt that the only possible way to perform a tracheotomy was to insert a bronchoscope and maintain the airway while the trachea was being found. On insertion of the bronchoscope through the paralyzed larynx, it was found that a fungating, ulcerating mass pro-

jected into the tracheal lumen at the level of the suprasternal notch. We were able to insert a 6 mm. bronchscope through the narrowed tracheal lumen. The incision was then made through the enlarged thyroid, and it was found that the trachea was displaced beneath the border of the left sternomastoid muscle. The carcinomatous thyroid had so involved the trachea that its walls could not be distinguished. In order to find the trachea, the bronchoscope was withdrawn partially, the lights in the room lowered, and trachea found by transillumination. An incision was made in the trachea, and a cane shaped Jackson's tracheotomic cannula inserted, passing below the level of the malignant involvment of the trachea.

CASE 2: A young man, 19 years of age, was referred from the medical service of Dr. Stengel of the University Hospital with marked dyspnea. The diagnosis was lymphosarcoma of the mediastinum. The case was not considered amenable to surgery, and, before X-ray treatment was carried out, it was advised that tracheotomy should be done. A lateral X-ray examination by Dr. Pancoast showed a large mediastinal tumor producing marked obstruction of the trachea. The mass extended upward from the anterior mediastinum into the neck, to the level of the cricoid cartilage. Preliminary to tracheotomy, a bronchoscope was inserted, and it was found that there was compression of the trachea almost obliterating its lumen from the level of the suprasternal notch down to the bifurcation. The left bronchus was also compressed. The bronchoscope was allowed to remain in position while the tissues overlying the trachea were separated; the tracheal rings were incised, the bronchoscope withdrawn to the level of the opening in the trachea, and a long, Jackson, cane shaped tracheotomy tube inserted, reaching down to the level of the bifurcation. In this way air was carried down to the right lung which was still functioning.

BRONCHOSCOPIC AID TO ESOPHAGOSCOPIC EVACUATION OF A RETROPHARYNGOESOPHAGEAL ABSCESS.

A boy, aged nine years, was admitted to the surgical service of Dr. E. L. Eliason at the University Hospital. One hour before admission, he had sustained a stab wound in the front of the neck caused by falling on a sharp portion of a

glass bottle. The sharp, pointed portion of glass had passed through the trachea and esophagus. His general condition was good, but there was present marked subcutaneous emphysema, which involved the neck and face up to the level of the zygoma and extended downward over the upper portion of the chest. There was a transverse incision in the front of the neck, just below the cricoid level, through which air passed on forced breathing. There was no dyspnea and no bleeding. Lateral X-ray examination of the neck showed no evidence of glass. Bronchoscopic examination showed a transverse wound in the anterior tracheal wall, just below the level of the cricoid, and a puncture wound of the party wall into the esophagus at the same level. No foreign body was found. The wound was dressed by packing the skin open, so that healing would take place from the trachea outward. The child's condition remained good until the third day, when his temperature rose to 104° F. There developed considerable difficulty in swallowing. The temperature remained high, the difficulty in swallowing increased, and there was marked dyspnea. On the fourth day, it was thought that the patient was developing a retropharyngeal abscess. On the fifth day following the accident, a roentgenogram by Dr. Pancoast showed an enormous retropharyngoesophageal abscess which was obstructing the larynx from above, and compressing the trachea below the level of the cricoid. On consultation, it was decided to evacuate the abscess esophagoscopically. It was deemed advisable to insert a bronchoscope prior to evacuation of the abscess, because of the tracheal compression and danger of aspiration of pus into the lung. On insertion of the bronchoscope, pus was found to be coming from the puncture wound on the posterior tracheal wall. The trachea was compressed so that its lumen was slit like down to the level of the suprasternal notch. Dyspnea was entirely relieved by the insertion of the bronchoscope. The abscess was then evacuated through the pharynx by means of the direct laryngoscope used as an esophageal speculum. The postpharyngeal wall was incised just above the cricopharyngeal fold, and an enormous amount of very foul pus and gas was evacuated, the infection being due to some gas producing organism, probably colon bacillus. Pus was aspirated from the abscess cavity and the pharynx until the field was clean. The bronchoscope was removed, pus being aspirated from the trachea, the dyspnea

was relieved, and the child returned to bed in good condition. The following day there was slight reaccumulation of pus, which was aspirated through the incision in the post-pharyngeal wall, using the direct laryngoscope for exposing the opening in the hypopharynx, the patient's head being held low over the edge of the table so as to prevent aspiration of pus into the lung. It was necessary to aspirate the pus in this manner as it reaccumulated on three successive days, after which time the abscess drained itself without further aspiration, and the boy's condition progressed to complete recovery. X-ray examination of the neck at the end of one week showed that no evidence of mediastinal infection or retro-pharyngeal abscess persisted. Recent bronchoscopic examination showed that only slight stenosis resulted from the tracheal injury. The child's condition was otherwise normal.

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DISCUSSION.

Dr. Austin A. Hayden, Chicago: In regard to the case in which the fistula was closed with abscess following, Dr. Tucker states that there was a great deal of mucus in the air passages at the time of operation. This operation was done under general anesthesia. I would like to ask Dr. Tucker whether or not intratracheal anesthesia with proper suction might not have avoided the latent abscess in this case.

I take it that Dr. Tucker's paper is of great interest to every man in this audience. It is of interest in two ways: To those who do bronchoscopic work it is of great interest to note the wonderful technic that has been developed by the Philadelphia Bronchoscopic Clinic. To those who do not do bronchoscopic work, the paper is of great interest in the way of prevention. I believe that most of these cases are preventable. The tooth case that he spoke of, the sponge case—certainly they were preventable—and most of them are preventable if the operation is done under general anesthesia and with the employment of proper suction apparatus.

I believe these two papers this afternoon, the one in regard to the audiometer and this one, indicate an extension of the field of otology. Dr. Jackson and his assistants in Philadelphia are pioneers in their work

—they are specialists in a specialty. I hope there will be more such papers before these meetings in the future.

Dr. Thomas E. Carmody, Denver: Dr. Tucker mentioned, in the first part of his paper, that formerly we considered endoscopy and esophagoscopy only in foreign body cases, but I am glad to see that we are getting into a new phase, a phase that is probably more important than the other.

The Doctor spoke of the esophagus as the cause of lung abscess. There are people who believe that some of this is due to hematogenous infection. I believe the work of Lynah and Myers has shown that practically all of these cases are due to the esophagus. Lynah found, that after tonsillectomy there was blood in the trachea in almost every case; and Myers has recently collected a number of cases, something like 200, of tonsillectomies in which he found blood in about 90 per cent of the cases. Even in cases where suction apparatus had been used, blood is found in the trachea and bronchi, and there is no reason why we should not have lung abscess.

The cases Doctor Tucker spoke of as massive clots in the lung we see infrequently, but, nevertheless, there are cases where the lungs and bronchi are clotted with blood secretions, cases that could be saved if bronchoscopy were done early.

This work should be carried out very frequently where it is not today, simply because a great many people believe it is too much of a major operation and do not try the simpler technic as used by Jackson and a number of others.

Dr. M. F. Arbuckle, St. Louis: There is a constantly growing interest in the study and treatment of disease in the lower air and food passages by direct methods. It is my feeling that this field is of equal importance and much wider scope than that which includes only the removal of foreign bodies.

It is undoubtedly a fact that much can be done with the class of cases just mentioned by the proper use of the bronchoscope. These patients are, of course, seriously ill, and great care must be exercised in the study of the case before doing bronchoscopy. Contrary to a rather widespread and unfortunate impression in the minds of the profession and the laity, this procedure can be carried out with little or no harm in suitable cases. In this connection, I think the bronchoscopist must be guided by the internist, whose duty it is to give a report of the patient's general condition.

It is possible by suction through the aid of the bronchoscope to remove infectious material from all bronchi large enough to admit a suction tube, thereby clearing the way for the entrance of air. This, of course, serves a two-fold purpose, namely, aeration and ventilation. In addition, the amount of absorption is very considerably reduced.

Repetition of the treatment is often necessary. But this is also true in the treatment of similar processes elsewhere in the body. In those cases of lung abscess in which the lesion is favorably located, there is little doubt as to the great benefit following bronchoscopic drainage. The change for the better is at times almost dramatic. In June of this year, I removed a peanut from the right main bronchus from a child, aged 7. Atelectasis of the middle and lower lobes was present before operation. Improvement followed for a few days, after which the fever again came up and the child did not do so well.

On bronchoscopic examination, it was found that the lumen of the main bronchus was occluded by swelling. By careful manipulation, the bronchoscope was introduced past the swelling, whereupon the lumen of the instrument was immediately filled with bloody pus, sufficient in quantity to drip from the outer end of the bronchoscope. By suction, all visible pus was removed and the patient was returned to bed. No further bronchoscopic treatment was required, and the patient has apparently made a complete recovery.

In those cases of deforming disease in or about the trachea requiring tracheotomy, insertion of the bronchoscope during the operation is of the greatest help and comfort to both the patient and the surgeon.

DR. SAMUEL IGLAUER, Cincinnati: Dr. Tucker has opened up a very interesting field, and I think those who are doing this work find that many internists now realize the value of the bronchoscope both as a diagnostic and a therapeutic aid.

In the two cases of massive collapse of the lung which Dr. Tucker presented, I think we can safely say there was an infected lumen of the involved bronchus; and had this block not been removed, the foreign substance in these lungs undoubtedly would have caused pneumonia.

Dr. King, my associate, recently had a case which must have been due to collapse of the lung. The child had been tracheotomized for tracheal diphtheria, and a few days later it was noticed that no air was entering the left lung. Upon the introduction of the bronchoscope, the left bronchus was found to be filled with a plug of inspissated mucus. After removing the plug on two occasions and ventilating the lung, the child recovered.

The occurrence of lung abscess after tonsillectomy, as Dr. Carmody has said, is undoubtedly due in many cases to the presence of blood and saliva. Myerson's work proves, I think, that blood is drawn into the bronchi, remains there and sets up an abscess. In Dr. Myerson's paper, he says that the reason some cases develop lung abscess is because of the faulty action of the ciliated epithelium. These patients with lung abscess probably had some previous disease process in the bronchial tree which has damaged the ciliated epithelium and prevented the expulsion of the inspissated blood.

The question of prevention has been mentioned. It is a simple matter, if you are going to leave a sponge in the tonsillar fossa, to tie a string around the sponge so it will not get away.

Transillumination in tracheotomy is a clever stunt, but ordinarily, when the bronchoscope is in the trachea, one can feel it and cut down on it without any trouble.

DR. EDWIN MCGINNIS, Chicago: We are always greatly indebted to Dr. Jackson and the Bronchoscopic Clinic for their exposition of what can be done in the way of treatment with the bronchoscope. I have just gotten through with two lung abscess cases following tonsillectomy. Last year, at the American Laryngological Society, I reported another case of lung abscess following tonsillectomy.

As to the pathology, and how this condition is brought about, I recall four cases operated externally at the Presbyterian Hospital, and there was great unanimity of opinion as to the findings at the time of operation. In those cases the lung was softened, and when the aspirating needle was put in, only a few drops of pus could be drawn out at any one place.

What I think about aspiration is this—saprophytes are inspired at the time of operation and blood gets down and walls them off, and it is a fertile field for that growth. With the bronchoscope, you can remove the plug and the lungs heal. As to the two cases I had recently, one had a single abscess in the right lower lobe; and the second case had three abscesses, one in the left upper lobe, one in the right lower lobe, and one in the right middle lobe. One bronchoscopic aspiration was sufficient to put these patients on the way to recovery.

Dr. George W. Boot, Chicago: It seems to me we might get a great deal of help in our work if we would remember the direction of the bronchi and trachea, and put the patient in the proper position to discharge the pus.

I think some of us will have to disagree with the Doctor, that a plug in the bronchus necessarily means pneumonia. The tuberculosis experts are in the habit of collapsing the lung for the cure of tuberculosis, and they do not report pneumonia from the operation.

One thing that is needed, is for the general practitioners and internists to take more care in diagnosing obstruction in lung disease. How often do we see cases where the patient is treated for tuberculosis, when the trouble is due to a foreign body in the lung which could be easily diagnosed if an X-ray had been taken.

I have here an X-ray of a young man who fell from a scaffolding and was unconscious. He afterwards developed a cough, and the doctor told him he had tuberculosis—they found the tubercle. He was said to have a positive Wassermann reaction, so the diagnosis was changed to syphilis of the lung. He went to one of the best clinics in the city and took salvarsan injections. Then he went to the City Hospital in order to get treatments free, and they took this X-ray picture of his lung, which showed a six-penny nail in the third division of the right bronchus.

TECHNIC OF JUGULAR BULB DRAINAGE FOLLOWING MASTODITIS WITH REPORT OF CASE. OPERATION. RECOVERY.

JOHN McCOY, M.D.

NEW YORK CITY.

Drainage of the jugular bulb is sometimes imperative, when we are confronted with a condition of suppuration in the bulb following mastoiditis and sinus thrombosis.

In approaching this surgical problem there are several important factors to be remembered.

1. The position of the bulb. It is deeply situated in the neck, directly under the floor of the middle ear, and in close juxtaposition to the facial nerve posteriorly and inferiorly.

2. The size of the bulb. In more than fifty percent of cases the bulb is very deficient or even absent on the left side.

3. The contents of the bulb. Besides the upper end of the jugular vein, it contains three large and important nerves which pass through it, the pneumogastric, the glossopharyngeal, and the spinal accessory. The spinal accessory usually being situated laterally, is most likely to be injured by manipulation when we reach the bulb.

Keeping these points in mind, there have been several methods advocated, each of which accomplishes the desired result with a varying amount of difficulty as a surgical procedure, and with a varying degree of safety to the surrounding structures, especially the facial nerve.

The simplest of these is the skin fistula method of ALEXANDER, in which the margin of the resected jugular vein is stitched to the surrounding wound, and in this way a direct path of drainage is established from the bulb.

Another method, devised by PIFFL, is as follows: After separating the membranous canal wall as we do in the radical mastoid operation, and pushing aside all tissues up to the Glaserian fissure, we remove the floor and part of the anterior wall of the bony external canal to the hypotympanum. Hav-

ing gotten this far, the upper portion of the internal jugular vein makes its appearance.

Voss' Method: After exposing the sinus and removing the bone over it, the lower part of the vein becomes more and more horizontal, the lateral wall of the sinus becoming the inferior. The lower wall of the sinus is followed by removing bone with gouges. By following the course of the vein with a probe, the distance of the anterior wall of the jugular fossa can be determined. Exposure of the bulb is accomplished by very cautiously removing the bony wall above the anterior boundary of the jugular fossa. (To avoid injury of the horizontal semicircular canal, the bone is not removed higher than one-half centimeter). The posterior membranous wall of the bulb is readily exposed in most cases where we have to deal with a well defined bulb. After the posterior wall has been removed up to the roof of the bulb, it sometimes becomes necessary to remove the edge of bone which marks the separation of the sinus proper and the bulb. This shows the sharp knee from where the lateral wall of the sinus goes into the posterior wall of the bulb.

Whiting's Method: By using the posterior semicircular canal as a guide, all the diploic bone immediately below it is removed, and in so doing the posterior aspect of the bulb itself frequently comes into view. Now taking as a guide the point of utmost curvature of the sinus posteriorly, he removes, in a direction downward and inward, the occipital groove and the jugular process of the occipital bone to a level which he considers is the lowest part of the sinus. He then removes the bone covering the bulb and knocks off the knuckle of the foramen lacerum posterior. This gives a splendid exposure of the bulb, and through this a catheter may be passed down through the portion of the jugular vein remaining in the neck.

Another method is that of Grunert's, otherwise known as the "gutter" operation, which is as follows: First a complete resection of the tip of the mastoid. Then separation of the three muscles from the base of the skull, namely, the sternomastoid, digastric and splenius capitis. The soft parts are retracted as we work toward the base of the skull in a horizontal direction, in order to reach the jugular vein. The operative field is sometimes considerably narrowed by a prominent transverse process of the atlas. This may have to be resected, and in

doing so one must be careful not to injure the vertebral artery. The rectus capitis lateralis is now pushed aside in order to remove the last bridge of bone between the free margin and the jugular foramen. The bulb is now split open from the sinus end. This concludes the methods of attacks by way of the mastoid.

There is another method of approach by way of the neck stressed by TANDLER. The mastoid incision and the incision for the jugular are converted into one. The sternomastoid muscle is separated for its entire length, and pushed posteriorly. We now search for the spinal accessory nerve. It is tied loosely with a suture, so that it may be in good view during the operation. We follow the course of this nerve to its exit at the jugular foramen. The finger is now passed deeply into the wound, and we find the space between the styloid process and the mastoid process. Here the exit of the facial nerve through the stylomastoid foramen may be exposed. The digastric muscle is separated from the digastric fossa and pushed forward and downward. If we work on a line below the stylomastoid foramen and this muscle, there will be no danger of injuring the facial nerve. After pushing aside the digastric muscle, the occipital artery may be ligated in two places and cut. The jugular vein may now be separated from the margin of the jugular foramen, which can be felt with the finger. Now loosen the uppermost portion of the vein, after pushing aside the periosteum at the base of the skull and the rectus capitis lateralis. The bone is now removed from the sigmoid sinus to the bony margin of the jugular foramen, and the sinus, bulb and vein are split wide open.

I believe that the most complete and well defined of the operations that approach the bulb from the mastoid cavity is Whiting's. This operation, however, is not possible in all cases, especially those where the mastoid process is short and deep, or where the sinus is far forward. I also believe that the operative approach by way of dissection in the neck is applicable in all cases, and gives the maximum of safety to the facial nerve.

Before resorting to the above described radical procedures, I believe in the following sequence: First, primary ligation of the internal jugular vein. Then should pus appear from the bulb later on, I proceed to a jugular resection, and through

the upper cut end of the vein, irrigation and drainage of the bulb. If this proves inadequate, incomplete or impossible, I then proceed as described above.

The following interesting case occurred on my service at the New York Eye and Ear Infirmary.

HISTORY. On March 30, 1925, Mrs. V. B., age 26, native of Finland, occupation housewife, entered the hospital with the typical history of acute inflammation of the left middle ear and mastoid for the preceding three weeks, and with all the indications for opening and draining the left mastoid.

OPERATION. On March 30, 1925, a simple mastoid operation was performed on the left mastoid by one of my assistant surgeons. The pathology found at operation was that the mastoid cortex was very dense. Pus was found in the antrum and tip cells. A smear from the pus showed streptococcus. The sinus was far forward. Some dura was exposed in the middle fossa. The wound was lightly packed and partly closed. The temperature at the time of operation was 99°. Four days after the operation, on April 3rd, the patient felt chilly and the temperature rose to 104 degrees. A blood culture taken at this time was negative. The blood count showed 4,800,000 red blood cells, 13,000 white blood cells and 83% polys. The temperature came down by lysis, and on the morning of the third day following was normal. That afternoon the temperature rose to 104°. In the absence of all other physical signs, the internal jugular vein was ligated at the level of the cricoid cartilage. The lateral sinus was then opened. It contained a clot and pure pus. Bleeding was established from the torqular end but not from the bulb. Following this operation, the temperature fluctuated daily, reaching a low point of 99° and a high point of 105°. At each dressing, a small amount of pus welled up from the jugular bulb into the lower end of the mastoid wound. Blood cultures were taken on April 4th, 6th, 10th and 11th. These were all negative. A blood culture taken on April 13th showed 36 colonies of streptococcus in 24 hours. We then decided to drain the bulb.

OPERATION JUGULAR BULB, April 14, 1925. Incision from the lower part of the mastoid wound to the point where the internal jugular vein had been ligated. The anterior edge of the sternocleidomastoid muscle was exposed and pushed back.

The gland over the carotid sheath at the angle of the jaw was removed, and the internal jugular vein was ligated above the facial vein. The internal jugular vein was now exposed up to the bulb, and a search was made for the spinal accessory nerve. In this case, however, it was not found lying on the dorsal surface of the vein, but was afterwards found ventral to the vein. The styloid process was located and the posterior belly of the digastric muscle was incised and thrown forward to cover the exit of the facial nerve at the base of the styloid process. The finger was then passed into the jugular foramen, and the vein was separated from the margin of the foramen. The bone was then removed from the sigmoid sinus to the bony margin of the jugular foramen, and the sinus, bulb and vein were split wide open. Following the bulb operation, the temperature did not rise above 102°, and blood cultures taken the following day proved negative. On the fourth day following the operation, the patient complained of a pain in the right shoulder. No swelling or redness was observed, but the temperature rose to 105°. On the following day, April 19th, the patient had a sharp pain in the left chest with cough, and spit up bright red blood. There were physical signs of a small consolidated area below the angle of the scapula behind. The temperature continued up and down for four days. The size of the consolidated spot appeared to remain the same. On the sixth day, there were definite signs of liquid in the pleura, and an exploratory puncture obtained a few c. c. of thin seropus. Culture of this showed hemolytic streptococcus. The wound in the neck was clean and healthy, so on April 26th she was transferred to the New York Hospital. Her subsequent history there showed that she had her chest aspirated three times, when an average of 500 c. c. of serosanguinolent fluid was removed on three successive days. On May 1st, the right shoulder was aspirated and pus obtained deep but extracapsular. On the same day, a thoracotomy was done under local anesthesia. The ninth rib was resected for a distance of 1½" in the posterior axillary line. Following these operations, the patient did well. Her temperature returned to normal, and she was discharged from the hospital, cured, on May 26th.

DISCUSSION.

Dr. Joseph C. Beck, Chicago: I have had the privilege of assisting in three such operations a man whom I do not think Dr. McCoy mentioned, Dr. Piffl, and his operation in these three cases was absolutely successful, with no facial paralysis. I have operated on one case of jugular bulb thrombosis. We have had a good many sinus thrombosis cases that we ligated, which developed metastatic abscess such as the one mentioned by Dr. McCoy, but did not operate on the jugular bulb.

The question is, when is such an operation indicated? The symptoms that we fear are general sepsis or infarction. Neumann, of Vienna, catheterized the jugular bulb, that is, he passed a soft rubber urethral catheter, and it was successful in many cases. I hope we may have data from eminent men in the literature, whose operative procedure has been successful.

The Doctor said it is easy to make an anastomosis of the facial nerve, but I do not agree with him. The knowledge of the location of that nerve and keeping it in sight while you are operating on the jugular bulb, will usually avoid trauma of the same with subsequent facial paralysis.

Dr. John McCoy, New York City (closing): I wish we could have such cases reported, because they are supposedly rare. In the experience of such men as Whiting of New York, he has seen altogether about five cases; the writer has seen three. I have no doubt they occur sporadically in the practice of many of the men here present, and I would like to hear from all who have had such cases—how they handle them and what is the termination.

ATYPICAL CASES OF ACUTE OTITIS MEDIA.

I. Case of Acute Otitis Media and Uremia.
II. Case of Acute Otitis Media Accompanied with Paralysis of the Facial Nerve.
III. Case of Acute Mastoiditis with Late Labyrinthitis

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Acute Otitis Media and Uremia.

The acute inflammations of the middle ear are characterized by greater or lesser active inflammatory symptoms (fever, pains), inflammatory hyperemia of the mucous membrane of the middle ear and of the ear drum, with a serous or bloody exudate, mucopurulent or purulent secretion in the middle ear cavity. This inflammation usually runs a typical course and subsides within a period of four weeks or thereabout. However, the otitis may run a typical course and complications may still develop within this four week period or thereafter. Within the first few hours following the onset of the inflammation in the middle ear, the patient may suffer from boring or cutting earache, and a rise in temperature. These symptoms are usually aggravated during the night. The pains often radiate over the side of the head, sometimes toward the eye or into the teeth.

As a result of this, the general condition of the patient suffers, his facial expression changes, he looks pale, his tongue becomes coated and he loses strength rapidly. Soon thereafter he develops some difficulty in hearing in the diseased ear, accompanied by annoying subjective noises or tinnitus. The severity of the disease, as well as its stormy or mild development, depend upon the type and the virulence of the infecting organism, and also upon the resistance of the patient. The streptococcus seems to be the most harmful infection. Sometimes, the periosteum of the mastoid just back of the attachment of the ear is sensitive to pressure as early

as on the second or third day. This acute irritation of the periosteum indicates an inflammation of the mucous membrane of those pneumatic spaces which are in close relation to the periosteum of the mastoid. There may result a swelling of the glands of the mastoid and of the large glands in the retromaxillary fossa, so they can be felt. Also a probably toxic irritation of the meninges close to the mastoid may develop, especially in children, namely when both ears are simultaneously involved. This meningeal irritation is manifested by somnolence, delirium, boring of the head into the pillows, and may be accompanied by vomiting and even general convulsions. All of these acute symptoms may subside, or may increase until the inflammatory secretion causes a spontaneous rupture of the ear drum, or evacuates through a paracentesis.

There are infections of such a high grade virulence, especially some of the steptococcic strains, which within the first few days of the disease of the middle ear invade the lymphatics or the blood stream or both, that they may lead to local or general complications.

The following is the report of a case of acute otitis media in a man, which within the first few days led to a rare complication, namely, an acute hemorrhagic nephritis with uremia.

The patient, a man aged 36 years, never before had any ear trouble. Following a severe coryza, he developed very acute pains in both ears. I was first called to see the patient during the evening of March 31, 1925, and found a very sick appearing man lying in bed, and obtained the following history from the family:

On January 26, 1925, seven weeks previous, the man was held up and shot through the right lung. He was cared for at Charity Hospital and dismissed as cured in February. A later examination of the hospital records revealed the fact, that during the period the patient was in bed, he could not urinate and was catheterized, but as soon as he was able to leave the bed he urinated naturally. Frequent urinalyses while in the hospital always showed the urine as normal. From this, we may conclude that a kidney infection or inflammation did not exist at this time.

When I first saw the patient, he was sick in bed for three days with an acute cold in his head, accompanied by violent pains in both ears and absolutely unbearable headaches. He

was suffering from dizziness, nausea, constant vomiting, a moderate nystagmus and temperature. Both ear drums were swollen, red, bulging, no details discernable. There was a slight serous transudate. Hearing in both ears seemed obtunded. The patient was dull and somewhat lethargic. Both mastoids were slightly sensitive on pressure.

Under cocain-adrenalin, a large paracentesis was made through both ear drums, evacuating creamy pus under pressure from both sides. Within a few minutes, the patient sat up in bed, acted somewhat brighter, said his pains were less and that he felt relieved.

On the following morning the general examination gave the following: Temperature normal, pulse 80, both mastoids very sensitive to pressure. A very mild, spontaneous nystagmus, no Kernig symptoms, hearing dull. During examination, violent vomiting. The patient complained of such violent headaches that he tossed around in bed, boring his head into the pillows. The nurse stated that he had not urinated, but may have passed urine during his bowel movements.

April 2nd. Very restless night. Continuous vomiting. Because his headaches were so very violent, his own house physician kept him under control with morphin. Both ears discharged thick, creamy pus. Patient passed a small quantity of bloody urine.

April 3rd. Temperature normal. Pulse 80. Profuse discharge of pus from both ears; continuous vomiting and unbearable headaches. On catheterization, 2½ ounces of urine were withdrawn, which appeared macroscopically to be mixed with blood. The patient's face and neck were definitely swollen and edematous.

At first the vomiting and unendurable headaches were considered as a beginning intracranial complication. However, when added to these symptoms there developed a suppression of the urine and edema of the face, the diagnosis was changed to one of uremia, and the patient was transferred to the Hospital Clinic, where Dr. A. S. Maschke found the following on physical examination:

April 4th: Slight edema around the eyes. Pinpoint pupils. No nystagmus. No rigidy of the neck. No Kernig. Pulse slow—pressure 140/180. Reflexes deep and superficial, all

positive. Headache is frontal. Nausea and vomiting. Urine sparse, presence of red cells in urine important. Disagreeable breath. Diagnosis: Uremia. Treatment: Sweats, Fisher's solution per rectum, also hot colon irrgations. Cereal diet. Keep patient warm at all times.

April 4th. Temperature normal. Pulse 84. Very thick, creamy secretion from both ears. Headaches and vomiting less violent. White count: 13,400. Urine: Albumin 4+; many red blood corpuscles, moderate number of pus cells, many coarse granular casts. Blood urea 60 milligrams per 100 c. c. According to the Folin-Youngsberry method, 10 to 15 milligrams per 100 c. c. is considered normal. Pus from the ear shows many long chains of streptococci. Intake 2210 c.c.; output 250 c. c.

April 5th. Temperature 36.6°. Pulse 68. Swelling around the eyes. Edema both legs. Headaches less violent. Vomiting has nearly ceased. Very profuse purulent discharge from both ears. Loud tinnitus. Hard of hearing in both ears. Blood Wassermann negative.

April 7th. Temperature 37.2.° Pulse 84. Vomited only once today. Headaches almost disappeared. General condition much improved. The ear discharge lessened.

April 9th: General improvement slight. Secretion from both ears. Mild headaches. No vomiting. Edema of the limbs gone, but edema of the face still present. Urine: Albumin, + 2. Sugar negative. Many red blood corpuscles, moderate number pus cells, many bacteria. Blood urea 42 milligrams per 100 c. c. blood. Esbach 2½ grms. in 24 hrs. specimen. Intake 1390 c. c., output 1240 c. c.

April 10-16th. General improvement in the kidney symptoms as well as the ear symptoms. April 16th. Only a slight discharge. No more vomiting and headaches. Tinnitus lessened: Urine: Albumin 1+. Moderate number of pus cells and red blood corpuscles. Esbach 2½ grms.

April 20th. Patient was permitted to sit in a chair. Temperature and pulse normal. Only a very slight secretion from both ears. Subjective finding very good. Urine: Albumin negative. Urinary output normal. Blood urea 16 milligrams per 100 c. c. which was now down to normal. April 21st. Patient was sent home from the hospital.

SUMMARY.

This case is one of a man, aged 36, who on the third day of his acute otitis media, even before the ear drums had reached a stage when they ruptured spontaneously, manifested an acute and severe kidney complication. This acute suppression of the urine seems to have no relation to the difficulty the patient experienced in urinating during the time he was in the hospital, seven weeks previously, suffering from the penetrating shot through his lung. During this time, the patient could not urinate while lying in bed, but could urinate when he was permitted to sit out of bed, and also the frequent urinalyses were always negative. The present acute suppression and uremia is simultaneous with, and seems to have resulted from the toxemia of the absorption of the virulent infection from the double otitis media, which did not begin to evacuate pus until after the paracentesis was performed on the third day. If we consider his present condition a toxemia resulting from a severe general infection originating in the middle ear, then we should consider two other contributing factors. First, that the ear drums did not rupture spontaneously, but that the pus was first evacuated on the third day after a double paracentesis; and secondly, that the patient's vitality must have been lowered by the wound he received seven weeks previous.

This patient, with an intracranial as well as severe general condition, improved gradually under conservative treatment, until he was restored to his former state of health.

CASE OF ACUTE OTITIS MEDIA ACCOMPANIED WITH PARALYSIS OF THE FACIAL NERVE.

A woman, aged fifty, consulted me at my office for the first time on December 26, 1924, with the history that for the past two days, following an acute cold in her head, she developed acute and severe pains in her right ear.

EXAMINATION. Temperature 37.5. Pulse 92. The entire right ear drum was very red and swollen, with definite bulging in the superior posterior quadrant. All details of the drum had disappeared. The mastoid was slightly swollen and very sensitive to pressure.

TREATMENT. A large paracentesis was made under local anesthesia, and was followed by a discharge of blood and

serum. The patient was put to bed and had the usual general treatment. Two days afterward, on December 28th, the discharge lessened, the pains increased and a second paracentesis was performed. The day following, there was a profuse purulent discharge, but with increased pain on pressure over the mastoid, and an impression of beginning facial paralysis. On the following day, January 1, 1925, which is the fifth day after the paracentesis and the ninth day from the beginning of any of her symptoms, she developed a complete paralysis of all the three branches of the facial nerve. The patient was transferred to the Hospital Clinic for immediate operation.

White count, 16,200. X-ray right mastoid: There was a definite diminution of the air content of all the cells, and especially that of the antrum. The intercellular septa were fewer in number than on the left side. There was no evidence of any destruction of the cortex. The posterior mastoid cells were very large and overlapped the sinus. X-ray left mastoid, normal. The X-ray findings indicated a mastoiditis on the right side, with partial destruction of the intercellular septa.

Operation. Under gas-ether anesthesia, the usual retroauricular mastoid incision was made through tissues which were somewhat edematous. The mastoid cortex appeared normal. On removing the outer layer of the cortex, thick, creamy pus welled out under pressure. The mastoid was of the pneumatic type, the cell walls partially intact, and the cells filled with pus and necrotic granulations. There were a large number of retrofacial necrotic cells filled with granulations; also, large necrotic periantral cells. When the antrum itself was opened, thick, creamy pus, under considerable tension welled out. A wide opening was made into the antrum. The cells covering the sinus as well as the tegmen tympani appeared normal. A light iodoform gauze drain was inserted and the wound was left wide open.

On the second day following the operation, there was an impression that the facial nerve was recovering, and on the fourth day, the patient could partially close her right eye. The drooping of the mouth was less apparent, and she could wrinkle her forehead slightly. The improvement was gradual and continuous. The patient left the hospital in two weeks, and at the end of the fourth week, the wound was completely healed, the ear was dry, and all three branches of the facial nerve fully recovered.

SUMMARY.

This is a case of a woman, aged fifty, who developed a complete paralysis of the facial nerve on the ninth day following an acute otitis, and on the fifth day following the paracentesis. The paralysis of the facial nerve was a definite indication for the immediate operation. The opening of the mastoid and the draining of the antrum seems to have made it possible for the facial nerve to recover itself.

The explanation for the development of the facial paralysis so early in a case of an acute otitis media is that it may be due to a collateral swelling or a toxemia of the facial nerve coming from the retrofacial cells, which were necrotic, filled with pus and granulations, and which lie just beneath the facial nerve and are separated from the nerve by only a thin layer of bone.

CASE OF ACUTE MASTOIDITIS WITH LATE LABYRINTHITIS.

A young woman, aged nineteen, consulted us at our office for the first time on Saturday afternoon, June 14, 1924, and stated that three weeks ago, following an acute attack of tonsillitis, she developed a severe pain in her right ear, followed by a spontaneous discharge of pus, since which time she was under the care of her family physician.

EXAMINATION. Left ear normal. Right ear: definite swelling over the entire mastoid and very sensitive to pressure. The outer ear canal normal. A profuse, creamy, pulsating discharge came from a perforation in the posterior-superior quadrant. Definite bulging of the posterior-superior canal wall.

FUNCTIONAL TESTS. Weber lateralized to the diseased ear; Rinne negative. Conversational voice heard about three feet.

SYMPTOMS. Severe headaches over the entire right half of the head. Also, severe pains deep within the ear. Temperature 37.5°. X-ray examination made at that time, and interpretation of the still wet plates revealed a clouding of all the mastoid cells. This clouding extended over the zygomatic cells. The cell walls seemed mostly intact.

IMPRESSION. This patient had an acute mastoiditis with possible complications, indicating an immediate operation.

She refused to go to the hospital until after Sunday. On

Monday afternoon, June 16th, the patient went to the Hospital Clinic for operation. At this time, the swelling over the mastoid had increased as well as the pressure pain symptoms. Leucocyte count 12,200. Temperature 37.5°. The operation was performed the same afternoon under gas-ether anesthesia.

Operation. The usual retroauricular incision was made through swollen and edematous tissues. The cortex of the mastoid appeared normal, and no fistula of the cortex was found. Upon removal of the first cortical layer, thick, creamy pus came out under pressure. Also, on opening the antrum itself, creamy pus again came out under pressure. The mastoid cells were necrotic, filled with pus and granulations, as well as the cells extending up to the zygomatic process. The tip of the mastoid was necrotic and was removed. The bone covering the sinus was necrotic, and there was a small perisinus abscess. The wall of the sinus was covered with granulations and thickened, and therefore the sinus was freely exposed above and below until it appeared normal, and the flow of blood through the sinus felt quite normal. There were cells posterior to the sinus, which were necrotic and filled with granulations. The tegmen tympani appeared normal. A light iodoform gauze packing was inserted and the wound left wide open. The temperature remained practically normal. On the fifth day, the packing was changed, and every second day thereafter. On the tenth day following the operation, a swelling developed in the zygomatic region, which gradually disappeared after five days. Three weeks after the operation, the patient left the hospital. The ear was dry, the mastoid wound healed and the functional tests good.

On the evening of July 16th, one week after leaving the hospital and exactly one month after the mastoid operation, I was called to the patient's home and told that during the night she developed a high fever, combined with very marked dizziness and violent vomiting, which she attributed to the eating of a large quantity of cherries before retiring. She was lying on her left, or healthy side, complaining of severe headache, dizziness and nausea. Her eyes showed a most violent spontaneous, horizontal rotary nystagmus of great amplitude and frequency to both right and left, and she had a definite past pointing to the right with both hands. No meningeal symptoms. Temperature 37.5°. Pulse 90.

IMPRESSION. Patient had developed acute labyrinthitis. She was immediately transferred back to the hospital, and an icecap put over the right side of her head and absolute rest ordered.

EXAMINATION, THE FOLLOWING MORNING. Temperature 37.2°. Pulse 84. Tongue moist. Violent headache. Marked nausea. No vomiting. Patient was lying on her left or healthy side. She said she could not lie on her right side because it made her dizzy and caused vomiting. No secretion from the middle ear. The drum normal. Slight sinking of the posterior-superior canal wall. A slight secretion through the mastoid wound. The region around the mastoid and zygoma sensitive on pressure. No meningeal symptoms.

FUNCTIONAL TESTS. Weber heard in both ears but more often referred to the left or well ear. With Bárány's noise apparatus in the left ear, loud spoken words were still heard by the right ear. The small, a-1 tuning fork could be heard by the right ear. The low C_{64} fork and the high C^4 fork were definitely heard.

EYES. Spontaneous horizontal and rotary nystagmus to both right and left of great amplitude and frequency. Typical past pointing toward the right with both hands. No caloric tests were made, in view of the fact that the patient unquestionably heard with her right ear. The patient was kept at absolute rest with an icebag over the right side of her head and mastoid.

July 18th. Temperature 37.1°. Pulse 96. Very restless night, yawning frequently. Continuous nausea but no vomiting. Moderate degree of headache. No discharge from the ear. Slightly increased serous discharge from the old mastoid wound. No meningeal symptoms.

FUNCTIONAL TESTS. Weber could not be definitely determined, but more often lateralized toward the well than the diseased ear. With a Bárány apparatus in the well ear, loud spoken or shouted words could not be heard by the diseased ear. The small a-1 fork was not heard. This gave the impression that the disease had progressed and resulted in a deafness in the right ear. This was sixty hours from the onset of the attack.

EYES. Horizontal-rotary nystagmus to both right and left, of lessened degree and frequency, somewhat stronger toward

the left than the right. Past pointing, with the right hand definitely to the right; with the left hand still toward the right, but in a lessened degree. The patient still preferred to lie on her left, or well side.

July 19th. Temperature 37.1°. Pulse 96. General condition improved. Headaches disappeared. The secretion from the mastoid wound practically ceased.

FUNCTIONAL TESTS. Weber could be heard only in left, or well ear. All tests pointed to absolute deafness in her right ear.

EYES. The nystagmus toward the left side of less intensity and less frequency. The nystagmus toward the right side absolutely gone. Past pointing with the left arm.

CALORIC REACTION. With the patient lying flat in bed, the right ear canal was irrigated with two quarts of cold water at 20° centigrade, without causing any increase of the existing nystagmus of the second degree to that of the third degree.

July 20th. Temperature 37°. Pulse 80. No headaches. Very slight nausea. Right ear absolutely deaf. Past pointing and nystagmus remained unchanged. Irrigation of the right ear with two quarts of warm water at 43° centigrade did not diminish the existing nystagmus to the left. From these findings, we concluded that the right labyrinth was without reaction to cold or warm water.

July 21st. Temperature 37°. Pulse 96. General condition good. Nausea ceased. The spontaneous nystagmus definitely weaker. First degree. July 23rd. Temperature 37°. Pulse 96. General condition good. Spontaneous nystagmus very weak.

July 24th-28th. During these four days, the patient's general condition improved and she felt comfortable as long as she remained lying on her left or well side. The nystagmus was lessened both as to frequency and degree. Past pointing of the right arm was still typical. The left arm no longer past pointed.

July 30th. Temperature 36°. Pulse 76. General condition normal. Nystagmus of the second degree toward the left side had lessened to that of the nystagmus of the first degree only. August 1st. Temperature 37°. Pulse 76. General condition good. Nystagmus lessened and at times did not exist. August 3rd. Past pointing with the right hand at

times only. Nystagmus toward the left only. August 7th. The patient was permitted to sit up for one hour. No reaction. August 8th. Patient out of bed. Very slight nystagmus toward the left side. August 9th. Nystagmus had entirely disappeared. The patient was transported home for convalescence.

Summary.

This case is one of a young lady without any previous ear involvement, who developed a tonsillitis followed by an acute otitis media with a spontaneous rupture of her right ear drum. After three weeks of purulent secretion from the ear, she developed a mastoiditis, which was operated. Ten days after the operation, there developed a slight swelling over the zygomatic region, which subsided after five days. From this time on, the patient made an uneventful recovery and left the hospital at the end of the third week with a dry ear and a healed operative wound, and good functional tests.

During the night of the seventh day after leaving the hospital, which was four weeks following the mastoid operation, she developed an acute fever with great dizziness followed by nausea and vomiting, which she discovered was increased by lying on her operated or right side. The examination revealed a dry ear, with slight serous discharge from the former mastoid wound. Her eyes revealed a violent horizontal-rotary nystagmus to both right and left, with very great dizziness, nausea, vomiting and past pointing with both arms toward the right. The hearing was still normal in the diseased ear. Under our observation, within a period of 48 to 60 hours, the patient developed absolute deafness and loss of the caloric reaction in her right ear.

The vital question for decision was now to determine what course to pursue, whether conservative or operative; if operative, it would mean opening of the labyrinth. According to Ruttin, in his books on Diseases of the Labyrinth, he says "shall we open the labyrinth or not in a case of diffuse labyrinthitis with complete loss of function?" In the beginning, we do not know whether we are dealing with a case of purulent or only with a case of serous labyrinthitis. Therefore, he advises waiting to see whether the symptoms become aggravated or whether they subside.

The differentiation between serous labyrinthitis as distinguished from a diffuse purulent labyrinthitis can be established only, if it can be established with certainty at all, by the functional tests. As long as any labyrinth function (hearing, caloric, or turning reaction) can be demonstrated, we must assume that the case is still a serous and not a purulent labyrinthitis, although Voss has described cases of serous induced labyrinthitis with complete loss of all functional reactions. In case of serous induced labyrinthitis, there follows a direct nonpurulent inflammation through the labyrinth wall; more often there is only a collateral edema. Ruttin states that "As a rule a labyrinthitis arising in the course of an acute otitis, as contrary to a chronic otitis, is more often only serous in type." In our case, we decided to await further developments. First, we were dealing with a case of labyrinthitis coming on as a late complication to an acute mastoiditis; and secondly, as the deafness became absolute and the caloric reaction disappeared in the diseased ear, the patient felt subjectively better. Her headaches had left, her dizziness was only very mild, her vomiting entirely gone, her nystagmus very much less, and there were no meningeal symptoms.

With absolute rest in bed and an icebag over her head, the labyrinth symptoms subsided gradually, until at the end of three weeks the patient left the hospital feeling well, free from all dizziness, nausea, vomiting or nystagmus, but absolutely deaf in the diseased ear.

Eight days later, ear tests were made with the following results: The right middle ear dry and the mastoid wound healed. Turning ten times to right and left, normal reaction. Caloric reaction of the right labyrinth had returned. Hearing on the right side absolutely gone. In this case, the auditory, which are the more sensitive fibers, were totally destroyed, while the vestibular fibers recovered. In confirmation of the diagnosis of serous induced labyrinthitis is the return of the caloric reaction.

Two months later, she again developed an acute tonsillitis followed by a discharge from the right ear, which subsided in a few days. Under local anesthesia, the tonsils were enucleated without any special reaction, and she left the hospital after two days. Periodic examination of the patient up to October, 1925, fifteen months after her operation, reveals an

absolute deafness of the right ear, with normal labyrinthic reactions. The ear is dry and the wound healed.

For the above reasons, we conclude that this was a case of serous induced labyrinthitis following an acute otitis and simple mastoid operation, which recovered as to life under conservative treatment, excepting for the resulting absolute deafness in the right ear, and after fifteen months manifests no further intracranial complications.

We wish to draw attention to a well known symptom which the patient manifested, and which is seen in many patients who develop an acute labyrinthitis, namely the "forced position" taken by the patients for their comfort. The patient prefers to lie on the side of the good or well ear. This corresponds to the quick component of the nystagmus. For example, a patient with a right labyrinthitis lies by preference upon his left side. This is, according to Ruttin, manifestly to be explained by the fact that in this position the patient reduces his field of vision, and thereby lessens the extent of the apparent movements.

DISCUSSION.

DR. ROBERT SONNENSCHEIN, Chicago: Just as in most affairs of life we are likely to learn more lessons from our failures than from our successes, so it is that from the atypical and unusual cases to which our attention is called, we gain much information as to important signs and symptoms of diseases.

Because of the anatomy of the middle ear and its proximity to the brain, lateral sinus, facial nerve, and the inner ear, infection of the tympanic cavity is fraught with many complications and dangers. While the acute suppurative process most often involves the structures mentioned by direct extension, it is possible to have symptoms of a systemic character, with the entrance of bacteria or their toxins into the general blood stream, with localization in various organs in the body. The three interesting cases presented by Dr. Metzenbaum illustrate extension to and involvement of the facial nerve, the internal ear (labyrinthitis) and one affecting the kidneys with consequent uremic symptoms.

Some months ago, I had occasion to see a young girl, 14 years of age, who had an acute otitis media in which a paracentesis was done in the first 24 hours. Despite adequate drainage from the ear, she had considerable fever and complained of severe pain in, behind and below the ear. It was very difficult, because of the free drainage, to explain the persistence of the pain. Suddenly, terrific headaches appeared about the 7th day, the patient became delirious and was transferred to the hospital. She developed all the symptoms of uremia, with suppression of urine, coma, and generalized convulsions. This condition lasted for several days and then gradually improved, the fever subsiding and the

urine becoming normal in about ten days. Suppuration from the ear continued for about three weeks, after which time the hearing reappeared, the ear was dry, and became quite normal. As I recall it, the pus in this instance showed streptococcus hemolyticus, and it was the severe toxemia, we believe, which caused the acute nephritis and uremia.

With reference to the facial paralysis in the course of an acute otitis media, let me say that this occurs not so very infrequently. Some of these cases we have operated and some we have not. It is true, that the onset of the facial paralysis in the course of an acute otitis media is regarded by many as an urgent indication for operation, irrespective of the presence of other indications for mastoidectomy, such as high temperature, sagging of the posterior meatal wall, tenderness over the mastoid, severe pain about the mastoid region, etc., etc. In one case, a boy about eight years old developed facial paralysis on the 7th day of an acute otitis media occuring early in the course of a scarlatina. We operated the same day and found a badly infected mastoid. After that, there was gradual improvement in the facial muscles, with return of complete function in about five weeks. On the other hand, we have seen several cases, in which for some reason or another operation was not performed, and yet there was fairly prompt, spontaneous recovery of function. Whether the paralysis is caused by direct pressure of the pus on the facial nerve because of the dehiscence of the Fallopian canal, either congenial or due to erosions from the pus, or whether, as Dr. Metzenbaum has indicated in his case, there was edema of the nerve due to inflammatory reaction of the bony structures about the facial nerve, it is not possible to say. It seems to me, that especially where there are also other symptoms indicative of mastoiditis, if a facial paralysis occurs, operation is probably the safest procedure, as opening of the mastoid cells and promotion of drainage will relieve direct pressure if such there be, or will lead to a subsidence of the acute infectious process in the bone and its possible inflammatory reaction about the nerve.

The case which the essayist has described in which labyrinthitis occurred as a complication of an acute otitis media, raises the old question of the difficulty in differentiating between serous and suppurative labyrinthitis. When all functions of the labyrinth, both vestibular and cochlear, are apparently lost, a diffuse suppurative labyrinthitis is usually assumed to be present. Indications for the labyrinth operation differ with various authorities. If the patient seems to be improving, or at least does not get worse, expectant treatment often serves well, as shown in Dr. Metzenbaum's case. If, however, the temperature rises to any appreciable degree, plus the other enumerated findings, prompt opening and drainage are often indicated to prevent meningitis from extension by way of the internal auditory meatus.

I believe that we are much indebted to the essayist for presenting these interesting cases, and thereby pointing out some of the very important complications which may accompany an acute oritis media.

Dr. Harry Pollock, Chicago: I think we are indebted to Dr. Metzenbaum for bringing these complications to our attention—I was going to say unusual, but they are complications which we are always looking for and which we get quite frequently.

Regarding the case of facial paralysis, I will say that it is quite a usual complication of acute mastoid infection. During the Spring, we had four similar cases occuring very early in the course of the disease. One, a youg dental student, had an acute otitis. He had a spontaneous rupture of the drum on the second day, and on the fourth day had a practically complete facial paralysis on that side.

Regarding the treatment at the stage where you have facial paralysis, I was much surprised to hear in the paper just read, that they let these cases go by without operation, because, I think, if there is mastoiditis present in an acute otitis media with facial paralysis as a complication, operation should be done immediately, notwithstanding any other symptoms present at the time, whether temperature, pain, or what not. The facial nerve which is damaged by this infection, one could say would not remain permanently damaged, and while that is true, yet the most of these cases recover quickly following the unloading of the mastoid. In my opinion, the palsy is due to the edema or pressure, rather than to any toxic effect of the infection. The cases which we have are due as a rule to streptococcic infection, but nevertheless, I think the palsy is due to the edema of the nerve, and the quicker we can unload the mastoid, the better and quicker your results will be.

Year before last, we had a case of otitis media in a woman of about 45, in which there was indication for operation—in other words, a typical case—but owing to the objection of the family, we decided to treat her conservatively. After about six or eight weeks, the ear became dry and healed. About three weeks after the cessation of the discharge, this woman developed a facial palsy. There were no symptoms referable to the mastoid. Then, of course, we were puzzled to know what to do in this particular case. But we decided to treat it conservatively, and after about three months, the palsy got well. There is no question in my mind, that had we operated on this patient during the acute stage of the mastoiditis, she would not have had the palsy and would have recovered quickly. The other cases we had cleared up in a short time—they began to clear up within 24 hours after operation. In the case of the dental student, his paralysis was practically cured inside of a week.

In regard to the cases of labyrinthitis, I want to draw attention to one thing. Of course, we know in these acute cases, that it is not only difficult but absolutely impossible to say at the onset of the invasion whether you have a serous or a purulent labyrinthitis. We have been advised to wait 24 to 48 hours after the invasion, to see whether the patient was getting better or worse. But just recently in the Zeitschrift für Ohrenheilkunde, there appeared an article by Libensohn regarding the socalled blood corpuscle sinking test. He had had a large number of cases in which he was able to diagnose positively a suppurative invasion from a serous in this disease. According to this test—depending upon the sinking of the corpuscles in the solution when left to stand for a while—he has proven that he can make this differentiation. To me, that has been one of the greatest advances in otology if it is true, because we all have these cases of a beginning labyrinthitis following an acute case, and we hesitate whether to operate—in all these cases we have done and should do a simple mastoid, thus unloading the pressure in the

mastoid, and do not go into the labyrinth. In our experience, we have found the end results were very bad in those cases in which we operated early. In those cases in which we let Nature wall it off, where we were dealing with a serious labyrinthitis, the patients got well. I think, if this new test proves at all practical, we will have made a forward step in labyrinthine otology, for when we can differentiate between these cases, we will know better how to cope with them.

Dr. G. W. Mackenzie, Philadelphia: About three weeks ago Dr. Metzenbaum sent me a copy of his paper, with the invitation to discuss the case of acute mastoiditis with late labyrinthitis.

The first criticism to be offered in this case report is the manner in which the essayist records the spontaneous nystagmus. For instance, when the patient was first attacked with vertigo, on July 16th, it is recorded that she had horizontal rotatory nystagmus of great amplitude and frequency, both right and left. Unfortunately, it is not stated to which side the nystagmus was the more pronounced. If the nystagmus was more pronounced to the right than to the left side, it would indicate an irritative lesion of the right inner ear. On the other hand, if the nystagmus was more pronounced to the left side, it would indicate a destructive lesion of the right inner ear. There is nothing in the report of the spontaneous nystagmus to tell which type of lesion was present. Again, there is no record of there having been any attempt made to ascertain the turning or caloric test findings, which would materially help in telling the character of the lesion present in the inner ear. This data would have been valuable, especially where there had been neglect of details in the description of the spontaneous nystagmus. Fortunately, there is one thing left us in the description noted in the report, and that is: "The definite past pointing to the right with both hands," which corresponds to what one would expect to find in a case of spontaneous nystagmus to the right side. This, then, in the absence of more definite data would indicate the presence of an irritative lesion in the right labyrinth, providing the cerebellum is normal.

A further criticism of the observation of the spontaneous nystagmus is the neglect to note the character and direction of the spontaneous nystagmus, with the patient looking straight ahead. For, in this position of the eyes, the vestibular nystagmus is permitted to manifest itself in its purest form, unhampered by any extraneous influences. It has been well known for many years, that a vestibular nystagmus, independent of the manner of its production, is increased when the patient looks toward the direction of the nystagmus, by reason of the added influence of the physiologic nystagmus, which is manifested or tends to be manifested in all normal individuals; and it is decreased when the patient looks away from the direction of his nystagmus, by reason of the influence of the same physiologic nystagmus, which tends to take from or counteract the vestibular nystagmus. Furthermore, if one is to record the spontaneous nystagmus which is manifested only when the patient looks to one side or the other, instead of when looking straight ahead, it is well to know in advance whether the patient has any weakness in one or more of the extraocular eye muscles that are brought into play when the patient looks to one side or the other. Vertigo, if not nystagmus,

of eye muscle origin, it may be recalled, was referred to yesterday by Dr. Foster Kennedy.

This criticism is directed not so much toward the essayist, as it is toward those who originated the idea of recording the spontanous nystagmic findings in the side positions. I am referring to Bárány and a few of his earlier followers. Another unfortunate inaccuracy has been developed as a result of not taking into consideration the normal physiologic nystagmus, and its influence upon vestibular nystagmus when this physiologic element is allowed to play a part. For instance, we hear of nystagmus of the first, second and third degree. The essayist also makes use of these terms. A far more accurate method for observing the intensity of the nystagmus is with the patient looking straight ahead, at a distant object, eliminating thereby, as far as possible, all extraneous influences. The intensity is then recorded as follows: The length of excursions at the equator of the globe, expessed in millimeters, multiplied by 1 over the number of seconds interval between the eye movements. For instance, an excursion of 2 millimeters length at the equator, occurring once every second, is equal to 2 times 1 over 1, or 2. The intensity of 1 millimeter length of excursion at the equator of the eyeball, occurring every half-second, is equal to 1 times 1 over ½, or 2, and these, again, would be equal to one-half the intensity of a nystagmus of 2 millimeters excursion length at the equator, occurring every two seconds, etc.

At a later date, July 18th, the essayist tells us that the patient had horizontal rotatory nystagmus to both right and left, of lessened degree and frequency, somewhat stronger toward the left than the right. This nystagmus would speak for a destructive lesion on the right side, in contrast with the irritative lesion, which the patient manifested earlier. Again, with the Bárány apparatus in the well ear, shouted words could not be heard in the diseased ear.

On the 19th of July, the nystagmus toward the right side, the essayist states, is absolutely gone. This, taken together with the observation on the 18th, that the horizontal nystagmus is stronger toward the left side, would suggest a destructive lesion in the right inner ear, which was confirmed by the absence of reaction to irrigation with cold water. It would be interesting to know the exact position of the patient's head during the caloric test, for the reason that, as commonly practiced, with the patient's head erect, the cold water produces a rotary nystagmus away from the irrigated side, and since the patient with a destructive labyrinthitis already has a spontaneous nystagmus away from the diseased side, we are left to judge whether the already existing nystagmus is increased or not; whereas, if the test is made with the patient's head inclined to the side, so that the irrigated ear is directed toward the ceiling, the presence of a positive reaction is indicated by the development of a horizontal nystagmus toward the same side. This makes the detection of a positive reaction a much simpler matter than with the head in the erect position, as commonly practiced.

As to the concluding remarks of the paper, I wish to endorse largely what the essayist has to say about the differentiation of serous labyrinthitis and purulent labyrinthitis. I doubt very much that serous laby-

rinthitis ever exists in the same *diffuse* form as suppurative labyrinthitis. If it does, it exists only as a very briefly lasting forerunner of the true suppurative form. What occurs in practically all cases of supposed serous labyrinthitis is a circumscribed destructive process, such as occurred in this case, and not a diffuse one. In other words, the case appeals to us as one of circumscribed destructive labyrinthitis, involving the cochlea; whilst, in the nonacoustic labyrinth, there existed a transient, congestive process, which ordinarily tends to recovery with slight, if any, impairment of function as in this case.

The case is an interesting one, and with the exception of the points raised regarding some of the technic, it is well reported.

Dr. Myron Metzenbaum, Cleveland, Ohio, (closing): My first two discussers supported me very well, and I think Mr. Mackenzie and I have no disagreement at all on the subject, when I tell him that all the tests for nystagmus were made with the patient lying in bed on a flat pillow, and asking her to look straight at the ceiling.

To enter into a final discussion of these questions at this meeting would take too long, but I believe our differences are those of technic rather than of the fundamental form of examination. The caloric reaction was made within 24 hours after the patient was first seen.

SOME REFLECTIONS ON THE MAXILLARY SINUS.

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The Dangers Connected with Needle Puncture.

It is now common knowledge that a number of deaths (something under ten) have been reported and attributed to this procedure. It is also well known that the alarming symptoms in these cases always followed the preliminary injection of air. In each case, so far as we are able to ascertain, the air was forced in under heavy pressure, the untoward symptoms immediately following, and death occurring in from a few seconds to twelve hours. The cause of death was attributed to an air embolus lodging in the brain. This explanation always seemed to me to lack something, to be unconvincing, and far from satisfying to any one who is naturally honest in his scepticism. In the first place, when one recalls the experiments on animals, where varying quantities of air were injected into the veins which were followed by no symptoms, it is hard to reconcile this as being the real cause of death; furthermore, as postmortem evidences in the brain are practically nil, positive deductions, so far as I can learn, have never been concluded at the autopsy.

In discussing this subject, let it be understood that I have never seen an actual case, so anything I might advance must be purely theoretic. Nevertheless, I have been intensely interested in the subject (being an inveterate user of the needle puncture) and have talked with many of those who have been so unfortunate as to have had one of these tragedies enacted in their presence. It would seem that all are agreed that obstruction was offered to the air at the first attempt to make an injection. To overcome this, more force was applied to the piston when suddenly the air gushed in, the patient moaned and fell over onto the floor, usually unconscious, with cardiac and respiratory disturbances, the condition gradually passing away, but on the other hand not infrequently ending in death. To my mind, much more than a cerebral air

embolus has occurred in these cases, for in antrum puncture this can occur only where the mucosa has been forced away from the underlying bone by the point of the needle, and the air can find its way only into the smaller veins, therefore no great quantity of air could have entered the vascular system from this source, hardly enough to cause an embolism. The symptoms of irregular heart, giddiness, dyspnea and cyanosis may be due to some vagus disturbance through irritation of the second branch of the trigeminal. Cocain, it must also be remembered, excites similar symptoms.

The point of the needle could not have been in the lumen of the sinus when the injection was attempted, else the air would have freely entered. Therefore, it was either buried in the swollen and inflamed mucosa, or the point had pushed it away from the underlying bone and had failed to penetrate through the membrane. The sudden bursting through of the air caused a nervous shock which, in some manner, the modus operandi of which I have attempted to explain, acted upon the cardiac and even the respiratory centers, with more or less serious results according to that particular case. In other words, the condition to my mind, represents more of a shock than the symptomatology of a cerebral air embolus. Pneumatic pressure, as we all know, acts much more suddenly than hydraulic, and this fact could easily explain why no one has ever seen these sudden collapses following the injection of aqueous solutions. Furthermore, if only a small quantity of air became imprisoned in one of the terminal vessels in the brain, the hemaglobin in the blood would quickly absorb the oxygen in it, with a rapid disappearance of all symptoms. That this is not the usual occurrence is shown by those cases which have been reported, as the symptoms continued for several minutes, and in some cases as long as twelve hours, before the recovery or death supervened. The great moral that this teaches is—whenever resistence is offered to the air about to be injected, the attempt to force the air through should under no circumstances be continued, but an investigation made at once as to the cause of the obstruction. If then, despite all our efforts, some impediment still remains to the passage of air, it is wise to discontinue the attempt and make a large enough opening into the antrum through the inferior meatus, so that there will be sufficient room on all sides of the needle for the injected fluid to escape.

In an experience of twenty years extensive puncturing, I have never seen a single case that gave me one moment's anxiety. This can only be attributed to the care shown in making the injection.

Another point in which all my pet theories have been revolutionized is after a needle puncture, with particular reference to the appearance of the returning fluid as a criterion of the pathologic condition of the sinus mucosa. We have been brought up, as it were, on the teaching that the most important and trustworthy symptom of a sinusitis was pus somewhere in the nose, and once found it should be followed to its source in order to arrive at a correct diagnosis. The absence of pus, despite repeated examinations, was considered sufficient evidence that the sinus mucosa had not become infected; consequently was free from pathologic changes. This dictum applied doubly to the maxillary sinus so far as needle puncture and lavage were concerned. If pus did not appear in the washings, no further thought was given to that particular sinus, and the findings were considered negative, meaning that no infection was present, and the sinus was free from disease. This, I think, was universally accepted as the proper interpretation to place upon the importance of lavage so far as its diagnostic value was concerned. Personally speaking, this tenet had become so engrafted upon the diagnosis of maxillary sinusitis, that I thought it little short of actual heresy for one to even whisper the possibilities of any doubt being cast upon its infallibility.

Some years ago, however, it began to dawn upon me that despite these negative lavages, I was finding a considerable number of maxillary sinus infections, which, on operation, showed such changes in the mucosa as could have supervened only after months, if not years, of inflammatory activity; yet as I said, perhaps repeated lavage had never shown any purulent secretion. This, in my own mind, I explained away by reasoning that a spontaneous evacuation had just occurred prior to my puncture and lavage, as this had always been carried out during morning office hours, and these spontaneous evacuations, when occurring, always took place shortly after the patient had arisen. These exceptions, however, became more and more frequent, until my own faith became shaken, when one day, while making a lavage, a black

rubber pus basin was used instead of the usual white enameled variety. I immediately noted that the returning fluid was of a light straw color, but perfectly clear and transparent, a circumstance that was not apparent when the white basin was used. Operation showed that a high grade polypoid degeneration of the mucosa had taken place.

It is said that one must cite one hundred similar cases to establish a precedent, but this one case was enough to revolutionize all my previous conceptions and overthrow the doctrines which I had persistently clung to since my first introduction into sinuology. I saw at once that the old adage which had become almost sacred, to the effect that in the presence of a maxillary sinus infection, the Lichtwitz needle could invariably be depended upon to bring out pus, was now ready for the discard. I had already seen this same thing occur with the ethmoid labyrinth, when hyperplastic degeneration was finally recognized as a separate and distinct entity from the suppurative infections, and had I at that time been gifted with the slightest bit of intuition, I certainly would have recognized such a possibility taking place within the maxillary antrum, which, after all, is separated from a portion of the ethmoid only by the thinnest possible osseus partition. We have all long since observed the thin watery discharge coincident with the nasal polyps (hyperplastic ethmoiditis), but who among us considered that a similar condition might exist within the antrum giving the same symptomatology; and yet an acute observer, having a repetition of a hitherto undescribed symptom (the withdrawal of a straw colored fluid), would have certainly drawn hypothetic conclusions and then endeavored to verify them by any means at his command. Unfortunately, however, this fluid does not always appear to be available, for not infrequently the puncture is negative, while at other times, in the same case, as soon as the needle is introduced, the fluid begins to drop out before one even has time to adjust the connections with the syringe.

What then is the precise condition with which we are confronted? The exact condition is a polypoid degeneration of the mucosa of the maxillary antrum, without any change in the normal appearance of the nasal mucosa, even in the ethmoid region. The local symptoms will depend entirely upon the amount of hyperplastic changes which have occurred within the sinus cavity.

In the beginning, and in the early part of the infection, I believe the symptoms are so light as to be adjudged a mild cold, rather chronic to be sure, or a slight vasomotor disturbance such as one would expect from trifling irritation.

As the hyperplastic area spreads, the symptoms, such as serous discharge, occlusion of nostril and sometimes sneezing, become more pronounced, often simulating mild attacks of hay fever. I think this will explain that hypothesis advanced some twenty years ago by the late Schadle of St. Paul, in which he stated that hay fever was due to antral disease, and reported a number of cures by lavage of the sinus. As we see it now, his cases must have been this form of antral infection.

When this hyperplasia becomes well advanced, polypi begin to show themselves in the nasal cavities, and just as in hyperplastic ethmoiditis, may give rise to recurrent polyposis of middle nasal passage. Indeed Hirsch[1], in a recent publication, goes so far as to state that he believes this form of disease the most frequent cause of recurrent nasal polypi.

The diagnosis in the early stages, when only symptoms of a mild irritation are present, can be aided by properly made roentgenograms taken in the anterior-posterior position, where the two sides may be compared. The sinuologist should train himsself in the interpretation of these exposures.

In the later stages, the symptoms will be so apparent that no further aid will be required, if one keeps in mind the possibility of this hyperplastic condition being present.

And so in summing up, one must remember that the dangers of needle puncture are largely a personal equation with the operator himself, which by the application of proper care and patience may be avoided. Regarding the incident of simple maxillary polyposis, I am not clear, but in any event, let us remember when we encounter those obscure cases of socalled vasomotor rhinitis, rose colds and the like, that we may be dealing with a case, the pathology of which is hyperplasia of the mucosa of the maxillary sinus.

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DISCUSSION.

Dr. W. P. Wherry, Omaha, Nebraska: Anything that emenates from Dr. Skillern, tempered as it is with experience and a sincere honesty, must be carefully considered and should be appreciated.

Although I do not use the actual needle in puncture, I do practise routinely the use of the curved trocar, probably the size of a match stick, feeling that the information gained by this means no inconsiderable part of my effort toward a diagnosis. Thus far I have had no untoward effects. If the odium of empiricism can be applied to puncture and irrigation, personally I would assign this to the negative place.

I am thoroughly in accord with the basic principle, that a negative washing does not make the diagnosis. In this connection, my reaction has always been—born of experience perhaps—that careful study of the results of this procedure is just as necessary as is that of a careful observation following any other surgical procedure.

Relative to the nonsuppurative, hyperplastic type of chronic maxillary sinusitis, I rather expect a more definite and annoying symptom complex than in the more frank suppurative type.

As a matter of routine, I insure antral drainage through an opening under the inferior turbinate at the time an extensive ethmoid lesion is exenterated, and in this connection, not only from my own practice, but in cases operated previously by other hands, I have observed where a subsequent Caldwell-Luc becomes necessary a polypoid degeneration at this intranasal site, or the area on the lateral wall directly opposite this created opening. I am of the opinion that this pathologic condition has been superinduced by fragments of bone carelessly left on the mesial wall, or by the repeated trauma to the mucosa from rough manipulation of the irrigating canula, all of which develops the thought, that regardless of the method used, care in the manipulation is of greater importance than the choice of the method.

Dr. Joseph Beck, Chicago: The subject that Dr. Skillern has presented I am sure must be of importance to all of us because these accidents, antrum puncture followed by serious complications as well as death, are of comparatively recent report—that is, from the time when the advice has been given that air should be used as a diagnostic measure—compressed air forced through a trocar. I personally have never indulged in such a method of diagnosis, although I use air in the reverse manner in attempting to draw secretions out, which is sufficient to make a diagnosis if there is pus in the antrum. I have seen two cases of complication from puncture; one when air was used, and the other where air was used also, but it was claimed that it was when they were using water for washing that the collapse occurred.

Let me cite this case. It has been recorded in the Transactions of the Chicago Otological Society (1924). The wife of a physician had an acute antrum disease with antrum accumulations. One of our local specialists was consulted and washed the antrum out through the natural opening, obtaining pus and also relief for the patient. A few days later, this patient had similar symptoms of accumulation of material within the antrum. This specialist had gone away from the city, but another equally efficient rhinologist was consulted, and he repeated this procedure

of washing out the antrum through the natural opening. Immediately afterwards, the patient collapsed, became unconscious, was taken home in an unconscious state. I saw her about eight hours after the accident, at her home, when she exhibited a most peculiar symptomatology, namely, a jerking of the head, conjugate deviation of the eye on the opposite side, a traction of the right arm and right leg. The opposite arm and leg were paralyzed. A neurologist and a medical man had been on the case before I came. I inserted a large trocar into the right antrum by way of the inferior meatus and withdrew a large quanity of straw colored bloody fluid, which had no doubt been used in the washing. It was reported that none came back in the washing before the patient collapsed. This patient had a very stormy course following this accident, but I am glad to report that she is now, several months afterwards, practically well.

In regard to the second question that Dr. Skillern brings up, namely, polypi within the antrum, it is most important, but I was a little surprised that he did not bring up the study of the literature of the pathology of nonsuppurative sinus disease. He quotes Hirsch as the man who recently called attention to this condition, but it was Uffenorde who many years ago described this condition in the German literature, and Skillern himself championed it in this country. For a long time it was argued back and forth, but recently Hirsch of Vienna has dwelt upon this condition of nonsuppuration and polyp formation in the antrum. It is interesting to know that Hirsch found polypi very frequently in doing sublabial operations. So, if we really want to cure these cases of polypi of the nose, we must not only clean out the ethmoid labyrinth, but really get to the antrum as well.

I want to call attention to another thing in regard to antrum puncture that has not been mentioned, namely, traumatic neuritis of the infraorbital nerve and hematoma of the antrum. This occurred in a case that has been the bane of my life and also of Dr. Norval Pierce, a man who had his antrum punctured by an associate of Dr. Pierce as a diagnostic measure. The trocar undoubtedly hit the infraorbital nerve. When we opened the antrum, we found a real hematoma of the lining membrane of the antrum. But worse than all this was the constant neuralgic pain of which the patient complained. I operated on the man three times for this constant pain in the face, believing he might have a nerve inclusion in the scar; the first time I operated also to stop the constant bloody oozing from the antrum. The latter condition was brought to a standstill, but the pain in the face was relieved only a short time by sublabial operation, returning much more severely. The man is a highly neurotic individual.

Dr. Cullen F. Welty, San Francisco: I happened to see one patient die where an attempted puncture of the antrum of Highmore was made. This was by Dr. Hajek, while in Vienna. Of course consternation reigned among the students at that time, as to the possible cause of this particular death. As you know, everything is posted there, and it was found the patient had a cerebral hemorrhage.

I think most of these cases are accidents, the deaths depending on something else. I have no explanation. I have seen air introduced into the cheek through the antrum of Highmore by puncturing the other side. I have seen emphysema induced by the Eustachian catheter, and I have seen water introduced outside the antrum with no bad results.

In regard to washing, I like what Dr. Skillern has to say, but I am not entirely satisfied. I have not interpreted it that way. Washing has been satisfactory to me. When we get this exudate the Doctor speaks of —this gelatinous mucus—I have interpreted it as an inflammation of some kind that comes from above, or an acute inflammation that will subside after washing a few times. That has been my experience.

Dr. John N. Coghlan, Portland, Oregon: About ten years ago, my associate, Dr. Davis, in washing an antrum had the patient suddenly fall over on his knees and die immediately. We had no autopsy on this case for some reason, which I think was a mistake. I know nothing more of that case, beyond the fact that Dr. Davis said he had used no cocain. He had washed the antrum while I was working in a little room next to him, and when he called me the patient was dead.

Two or three years ago, the same unfortunate accident happened to me. An internist sent a patient to me about the noon hour, when I was a little exhausted. I immediately cocainized with a 10 per cent. solution, washed the antrum and found absolutely nothing, and then, absentmindedly, I took up the compressed air (which is not my habit) and pumped in air with a pressure of 15 to 20 pounds. After I withdrew the needle, the patient said "I feel badly." She did not look pale, but in a minute or two I noticed she had practically gone out. I called for aid, and we restored her again after artificial respiration of about five minutes duration. She came around nicely, and we moved her to a couch, but she died in a few minutes. We had a thorough autopsy, but it proved nothing. I was of course exonerated of blame in connection with the death, but I still think it was due to the air which I blew into the cavity.

About two years ago, a little boy came in with a lot of secretion in the nose, and I decided to wash out his antra. He was a little bit unmanageable, but I cocainized him and washed one antrum, and then took the Politzer bag to dry it. There was a good deal of difficulty in getting the air through, and suddenly his eyes rolled up and he said, "I see things upside down." A peculiar pallor spread over his face and I was almost paralyzed, but in a few minutes he was all right. I do not know what happened, but I did something which caused this diplopia and the change in the color of the skin of his face. It was undoubtedly the compressed air, and I congratulate myself that he did not die.

Dr. Samuel R. Skillern, Philadelphia: I wish to bring before the Academy what I believe to be a new treatment in purulent conditions of the accessory sinuses and middle ear, namely, the intravenous injection of triple distilled water. As I have had my best results in chronic purulent maxillary sinuitis cases which have not responded to the usual treatment, I speak of it under the discussion of my brother's paper. If this treatment gives the results it promises, as it has in the comparatively few cases I have tried it on, it means less radical operating and the relief of those purulent cases of sinuitis that so stubbornly resist treatment. Surgery of course is essential, for without good drainage this treatment is of no avail. I would like to give you the history of two cases.

The first was a young French woman who had been a German prisoner for the duration of the War. She had been made to live in a cellar under the most unhygienic conditions. During the winter of 1914, she

contracted influenza and was sick for six months. During her convalescence, her nasal condition developed. I saw her first in January of this year. Needle puncture of the right maxillary sinus showed thick brownish-yellow pus. The left was filled with a very fetid, dirty brownish, semifluid pustular discharge, which mixed intimately with the normal saline solution used for the irrigations. I advised a double Caldwell-Luc, but as she was a school teacher she could not take the time off until vacation. The weekly irrigations relieved her somewhat, and what was most important, relieved the very fetid odor from her breath. A colleague of mine told me of the success he was having in neuritis with the injection of triple distilled water in the vein. I was very skeptical, but decided to try it for this condition. To my great gratification, my patient was greatly improved after the third injection. Continuing the biweekly irrigations and the injection of distilled water, I found, after the ninth injection, that her right antrum was free of pus and the character of the pus in the left antrum had changed from a fetid liquid to a golden globular mass. The condition of general health was greatly improved and all halitosis had disappeared. Her condition remained more or less stationary until late in March. By stationary, I mean I could not seem to get rid of a small quantity of pus which presented in her left maxillary sinus, until the twenty-third injection of water had been made. I saw this case just before I came to Chicago, and she states that she has had no nasal condition since I last saw her. Washings from both maxillary sinuses returned clear.

The other case was also one of double maxillary sinuitis, who was being treated by a colleague. She had had biweekly washings of both sinuses for three months without improvement. After her first injection of triple distilled water, she told me she felt much better and had had no discharge for two days. The eighth injection found her right antrum clear, and the twelfth her nose and sinuses free from all pus.

In case any of you gentlemen would care to try this treatment, I will tell you the triple distilled water I use is made fresh every 48 hours, put up in sealed Venetian glass containers, and autoclaved to make sure of its sterility. One c. c. is given in the median basilic vein every fourth or fifth day. This dose is gradually increased until 3 c. c. are given. The dose is increased only until you feel you are getting a reaction. It is then held at that dose until your case is relieved. An overdose will increase the symptoms.

Dr. John A. Cavanaugh, Chicago: Dr. Beck has given you the history of a case in which it happened to be my misfortune to wash out the maxillary sinus. I would like to mention the technic with which this irrigation was done. A small pledget of cotton with 7 per cent solution of cocain, no adrenalin, was placed at the natural opening of the sinus and left there for a few moments. Pierce's canula was introduced at the natural opening. The canula slipped into the cavity without any effort. Water was used first (not air). The water met with resistance, so the syringe was removed and the canula attached to a small four ounce air bag and slight pressure exerted, which also met with resistance. The water syringe was reapplied, and when the water was reinjected, the patient threw up her hands and fell backward. She became unconscious. I thought it was an hysteric attack. The doctor and I worked over her for an hour

or more with but little benefit. There was a deviation of the eyes to the right side. The patient was unconscious for three days. Then she regained consciousness for about a week, and then became unconscious again. Her temperature ranged from 102 to 103. She had complete paralysis of one side with partial paralysis of the other side. The Doctor tells me today that the left side, which was the paralyzed side, still lags a little, otherwise the patient is in good condition.

I was especially glad that Dr. Skillern mentioned the fact that he does not believe air is responsible for these cases, because I do not see how air could be responsible in this particular instance. I wish Dr. Skillern would express himself in regard to this case. It was reported to the Chicago Laryngological and Otological Society, and will appear in their transactions.

Dr. B. N. Colver, Battle Creek, Michigan: We have had two deaths from needle puncture in our clinic. We have also had several cases where the symptoms varied from a very slight disturbance to even a brief paraplegia. In the winter of 1910-11, I had the opportunity of studying with Doctor Skillern and learned the technic of the Lichtwitz needle puncture. Until 1917, I did many cases with no unfortunate results. In 1917, one of my colleagues had irrigated one antrum, and in the irrigation of the other side, the patient had cardiac failure and died suddenly. In 1923, the second case occurred. I had washed this case four or five times before. This day I did the washing in the usual way, cardiac failure ensued and the patient had marked respiratory disturbance. She died about a week later.

In the cases that we have seen, it has appeared to us that the cause of the symptoms was a fifth nerve-vagus reflex. I could not understand why, and do not understand now, but I will give my surmise. It seems to me that the inner surface of the middle turbinate opposite the opening of the antrum is extremely sensitive, and when the air strikes against this area it sets up the reflex. If that is true, then cocainization of the middle turbinate would obviate the reflex. In the cases we have seen, there was not an obstruction of the ostia with high intrasinus air pressure, but rather a free rush of air out against the turbinate.

Dr. Noah Schoolman, Chicago: I wish to call attention to a test which might prove useful in hyperplastic condition of the antrum. I first learned it from Professor Glass of Vienna. It is the tuning fork test. If you put a vibrating tuning fork at the root of the nose, you will have a lateralization of sound to the ear on that side, if the antrum is filled by a hyperplastic material. Dr. Glass reported that about twelve years ago. Another man has recently written about this test, and adds, that if you put a vibrating tuning fork at the root of the nose, you will not only have a lateralization of sound to the ear, but you will feel a distinct vibration in that antrum as contrasted with the other. I mention this for what it is worth.

Dr. W. E. Grove, Milwaukee, Wis.: I believe the number of these cases is somewhat greater than Dr. Skillern reports. In 1922, I was able to collect 15 fatalities. A great many cases, as you will see from the reports made today, are not reported in the literature. I believe air embolism is at the basis of these deaths, for the following reason. In the first place, as Dr. Skillern remarked, the collapse occurs at the point when air is being forced in; second, the ordinary type of autopsy will not reveal air in the brain. The brain must be taken from the skull cap under water; and in

two such autopsies, one made in Canada and one in England, air was discovered in the vessels of the brain and in the vessels of the pia. In another case which recovered, but which had a similar collapse, the operators were able to feel the air passing out of the jugular vein. In another case of transient blindness following irrigation by air, air was discovered in the veins of the retina, and when this condition subsided, the vision immediately improved.

Whether this condition was due to air embolism or something else, I do not know, but certain it is that the introduction of air adds another element of danger, and we do not need the introduction of air to determine whether the needle is in the antrum or not.

Dr. Ross H. Skillern, Philadelphia, (closing): I think my old friend, Joe Beck, forgets some of the things that happened a long time ago. Before this Academy, fifteen yeasr ago, I read a paper on this subject, and was violently opposed at that time by the late Dr. Ballenger. The best support I had then was Joe Beck and now he gets up like Pontius Pilate and washes his hands of this man. Well, he cannot do that, because I have a friendship for him that nothing will ever sever.

He spoke about the controversy between Hirsch and Uffenorde. I happen to know something about that, because I was with Dr. Hirsch in Vienna when this discussion took place. He says he believes that all cases of recurring nasal polypi come from this hyperplastic condition of the antrum. I do not believe I would go that far, but he has, and now is working up another article along those lines.

Regarding Dr. Coghlan's report of two deaths following needle puncture, I must say that honest confession is good for the soul. When I said that I had never had a death from doing this, I am not assuming a holier-than-thou attitude at all. At one time I said I had never had a death following tonsillectomy, and I used to glory in it; but I do not do that any more. I have joined the other class. I have never seeen anything in the way of serious complications, but there is no telling when I may. That is my point of view, and will remain such until I have reason to change it, which I hope will not happen.

CONGENITAL OCCLUSION OF THE CHOANAE.

KENNETH A. PHELPS, M.D., F.A.C.S.

MINNEAPOLIS, MINN.

"The choanae are communicating passageways—not mere orifices—between the nasal fossae and the nasopharynx." They are very narrow at birth and infundibuliform in shape (Fig. 1). The osseous boundaries are: above, the alae of the mover; laterally, the median plates of the pterygoid process of the sphenoid; medially, the vomer; and below, the horizontal plates of the palate bones. The bony walls cause the normal nasopharyngal communications to be permanently open and free, unless blocked by some outside structures, such as adenoids.

The choanae may be closed congenitally. The occlusion may be membranous or bony, unilateral or bilateral, complete or incomplete. Richardson[1] classifies congenital occlusion of the choanae as follows:

(1) Membranous. This is formed posterior to the nasal cavities in the nasopharyngal cavity, but in contact with the postnasal orifices so as to completely obstruct them. The membrane extends from the upper surface of the soft palate, where the hard palate joins the postnasal cavity.

(2) Osseous. The bony plates are usually vertically placed—concave pharyngeal aspect—within the nasal chamber, a millimeter or more from the free border of the postnasal orifices.

(3) Developmental. The bones entering into the formation of the postnasal orifices become united, thus more or less completely obstructing them.

Congenital, complete, bilateral occlusion is very rarely observed, for the reason that most babies with this affliction die very shortly after birth. Mouret[2] affirms that he has been able to find but four authentic records of such babies who lived. I have one additional case to report today. Many such cases were reported as complete occlusion, it is true, but in his opinion, they were really incomplete ones.

Up to 1923, Libersone[3] found reported one hundred and seventy cases of all types. Probably McNaught[4] is correct when he maintains that this condition is very much more frequent than has been generally supposed. One reason for believing this, is that cases of unilateral occlusion are occasionally discovered in adults who did not suspect that they had any such trouble, or who were so little annoyed by it that they did not seek relief. Cases of bilateral occlusions have

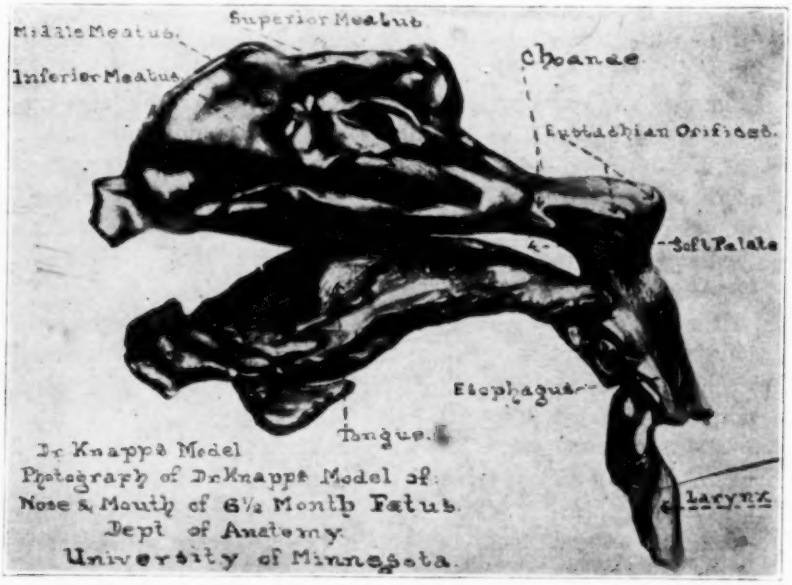

Fig 1. Metal cast of nose and mouth made by filling the cavities with metal and removing the surrounding tissue.

been discovered at 14 and 16 years of age. Cases of asphyxia neonatorum are at times caused by congenital occlusion of the choanae; occasionally this is recognized, but more often it goes undetected.

There are certain coexisting factors in connection with this condition that are very well worthy of mention. It is interesting to note that females are afflicted in this way fully twice as frequently as males, according to the best statistics that are obtainable upon the subject.

So far as we have been able to determine, it is not hereditary, although there is on record one family, six members of

which were affected. It consisted of a mother, her two sisters, her two daughters and a son.

Quite frequently it is associated with other deformities, although in most cases they are deformities which it may, perhaps, produce, such as high arched palate. At other times it is associated with other congenital defects—coloboma of the iris, for instance.

When it is unilateral, it more frequently occurs on the right side; but it occurs three times as often bilaterally as it does unilaterally. Ninety per cent of the cases are said to be of the complete, bony type.

The pathogenesis of this condition is very interesting, and there are various views upon the subject, some of which are conflicting. Jacques[5] reports a case which he believes was due to congenital lues. Trauma at birth was mentioned by Ullman[6], but he abandoned this view because it could be caused only by a fracture—and a fracture of the choanae at birth is practically impossible. Others believe that this is caused by a hyperplasia of the bony skeleton of the posterior nares, because of the finding of a small opening in the upper section of the choanae near the septum. Some investigators believe that the vertical plate of palate bone is the one that proliferates (Bitat, Kundrat, Bennett[7], Schroetter, Hopman[8], Boty, Ricardo and Chiari); whereas a different school maintains that the horizontal plate causes the trouble (Luska and others). Still a third school is of the opinion that it comes from an overgrowth of the vomer.

Among those who are of the opinion that it is caused by a persistent bucconasal membrane may be numbered Shaeffer[9], Bonly, Hoag, Hochheim[10] and Dusque. Ducuring thinks it is a deformity of the soft palate.

A review of the embryology, according to Schaeffer, may be enlightening. "The characteristic epithelium of the nasal areas is recognizable as early as the third week of embryonal life. During the fourth week, these areas become passively depressed by a positive increase in the thickness of the surrounding mesoderm, which pushes the overlying ectoderm into relief. The nasal pits are thus formed. In a thirty-five day embryo, the nasal pits have deepened sufficiently to partake of the nature of cleft like fossae, which end blindly at their dorsal and inferior termination. This is the primitive

nasal fossa, and its dorsal growth meets the ectoderm of the oral fossa and forms the bucconasal membrane. (Fig. 2.) This membrane is so attenuated in a thirty-five to thirty-eight millimeter embryo, that rupture usually ensues and the primitive choanae are formed, which establishes the communication between the nasal fossa and the oral cavity, and also delineates the primitive palate. Lack of this rupture leads to atresia of the choanae, as a result of an organization of the epithelial plugs which occasionally block the choanae; rarely osseous tissue develops in the organization.

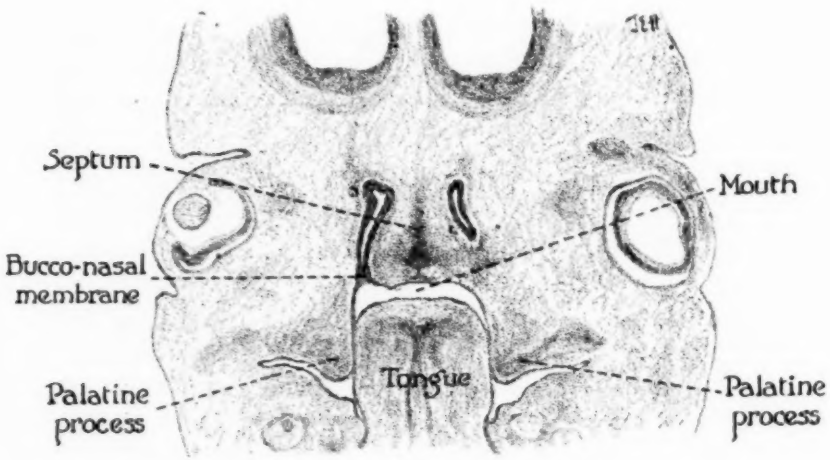

Fig. 2. 35 day human embryo. Bucconasal membrane not ruptured. (University of Minnesota—Laboratory of Anatomy).

These primitive choanae do not correspond in position to the definitive choanae, which are placed farther dorsal and are established in the third month, when the definitive palate is completed.

Ecker and His[11] do not concur with Schaeffer. It is their belief that as soon as the nose and the mouth are formed in the human fetus, there is a connection between them; that this connection does not put in an appearance at a later period of development. But they agree with Schaeffer that the nasal cavities later are partially shut off from the buccopharynx by the growth inwards of the hard and soft palate.

Hochstetter[12], Tiemann[13] and Keibel[14] are of the same opinion as Schaeffer, holding that in cases of choanal atresia,

it is possible for the nasobuccal membrane to become permeated by mesoderm, in which event it fails to break down.

Fraser[15] directs our attention to the fact that the theory of Schaeffer and those who concur with him is in line with our knowledge of the development of the vagina, anus and the external auditory meatus. He also affirms, that in the majority of cases of choanal atresia, the occluding membrane is well in front of the choanae, which lends support to the theory that congenital atresia is due to the failure of the nasobuccal membrane to break down.

Frazer[16] shows a very nice series of drawings showing the development of the septum and the posterior nares. These I have reproduced. (See page 358.)

You will note that the illustrations give support to the theory of Hopman, to the effect that the choanae may be narrow enough to produce a stenosis. But Hopman ascribes this narrowing to the presence of an asymmetric fetal anlage, and he reports a number of cases in support of his contention that need not be cited here.

Mouret[2] refers to Professor Vialleton, when he asserts that the two gutters appearing in the roof of the mouth, connecting the nose and the mouth, are improperly called the primitive choanae. Physiologically, they may be so, he grants, but not topographically or anatomically for the reason that the definitive choanae are further back, and lie in the opposite direction. Professor Vialleton further maintains, according to Mouret, that the nasal cavities owe their growth to a proliferation of epithelium. If this proliferaton does not progress in a normal manner, there occurs an occlusion of the choanae. He does not consider the persistence of the bucconasal membrane an etiologic factor in such cases.

We have seen that the authorities cited above are not in complete accord. It is the belief of the present writer that the cases that fall in group two, given above, are very likely caused by a persistent bucconasal membrane, because the occluding plate, or membrane, is well in front of the choanae. Those that are under group three are probably caused by narrowing of the choanal canal.

There are two other points that properly should be considered at this time. The first is that the persistence of the bucconasal membrane would not necessarily interfere with the fusion of the palatine processes. Accordingly, any de-

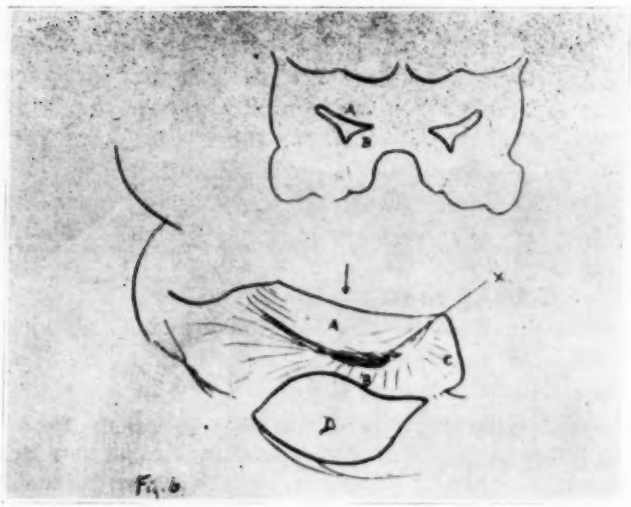

Fig. 6. Inner wall of nasal cavity at 5th week. Primitive palate (D). The nasal cavity (ABC) with large opening anteriorly and closed by the bucconasal membrane (indicated by dotted line). (A) is really the roof of the cavity as shown in the transverse outline above.

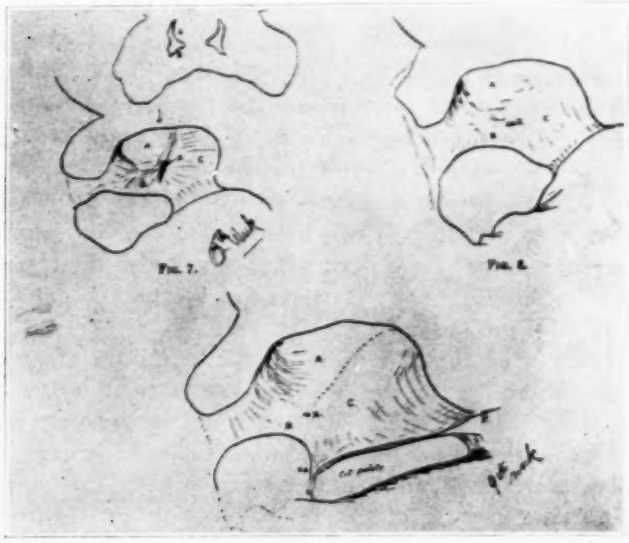

Fig. 7. Sixth week. Here the roof of the cavity (A) has swung around to become the side as shown in the transverse section. (C) has increased by the upward growth of the maxillary mass, and the posterior nares are much larger. The bucconasal membrane has ruptured.

Fig. 8. Eighth week. Showing still larger choanae (C).

Fig. 9. Ninth week. Showing how the large choanae of the last stage are nearly occluded by the fusion of the palate folds. Compare this with the model shown in Fig. 1.

formity of the palate is independent of the condition of the choanae. The second fact to which I desire to direct your attention, is that choanal occlusions, whether bilateral or unilateral, do not always, of necessity, produce a high arched palate[19]. In other words, high arched palates are not necessarily the result of mouth breathing.

The comparative anatomy of the choanae and the evolution of nasal breathing is of some bearing on the subject. (Negus[21]). In the majority of fish, there is found to be no communication between the simple nose and the food tract. In Dipnoi, however, such a communication does exist. In these fish, inspiration is affected by the swallowing of air through the mouth, as occurs in some Salamanders and Newts. While the air breathing proceeded, there would, therefore, be no circulation of air through the nose, and the olfactory sense would not be acute; therefore, a communication or choanae is present in the roof of the mouth, whereby air can be made to pass over the olfactory rosetti, so as to give off odor sufficient for preception of scent. In these cold blooded animals, there is no necessity for air to pass through the nose as far as respiration is concerned.

Thus air enters in very low types by the mouth, nasal breathing being a secondary modification of respiration, and as the path of evolution is traced up, there is to be found in all cases a continuation of this close relation of air tract and food passage. When nasal respiration is firmly established, there is still a necessity for the crossing of the two streams. In birds, Ingersoll[22] says, the posterior nares have finger-like projections which interlace and can shut off the nose from the pharynx and larynx.

The probable sequence of events in the evolution of nasal breathing is illustrated by the facts known of human embryology. The nasal area develops, the nasal pit appears, the bucconasal membrane breaks down and the palate is completed.

Congenital occlusion of the choanae was first recognized by Otto in 1830 at postmortem. In 1853, Emmert[17] reported the first known case in a living patient, a child of seven years of age with a double occlusion. He operated and the result was a complete recovery.

Symptoms of bilateral complete occlusions are very striking, and are so definite as to make the diagnosis an easy one

and quite apparent. This is not true of a unilateral or a partial obstruction. When a complete occlusion is present, the infant at birth has much difficulty in breathing. It is impossible for the baby to breathe through its nose, and it will not breathe through its mouth, because mouth breathing is an acquired habit. The tongue, being markedly developed for suckling purposes, increases the difficulty in mouth breathing. Accordingly, the mouth must be held open to relieve the difficulty.

Difficulty in nursing is always associated with the condition that we are discussing. It is necessary that the infant be fed with a spoon or by means of a stomach tube. As the infant grows older, it may partially overcome this symptom, but it is never able to take its entire feeding without difficulty, usually swallowing air with the food.

Another symptom is that the nasal cavities are always filled with a secretion of a peculiarly glairy, gelatinous consistency, which irritates the lips and the nostrils. If the baby is held face down, this secretion will run out. It is unable to clear its own nose.

Anosmia is another symptom that always accompanies this condition. If it is discovered in early life and proper treatment is instituted, the sense of smell will develop normally. In older people the sense of smell has returned upon removing the choanal obstruction.

Some observers have noted that when the patient weeps, tears run out of the nose. Others have observed diminished lung expansion on the side of the nasal obstruction, an elevation of blood pressure, incontinence of urine, dyspepsia, nasal tone of the voice, dry pharyngitis and inability to indulge in violent exercise.

Very little information concerning this condition can be obtained by examination of the nose of an infant by anterior rhinoscopy; but I find it comparatively easy to see the posterior nares through the mouth by means of a Holmes nasopharyngoscope. Another method is the use of a palpating finger in the nasopharynx. It is impossible to pass a probe through the nose, which is very convincing proof of a posterior occlusion. Even when the procedures outlined above are followed, one cannot be absolutely certain of an occlusion without ruling out adenoids, hypertrophied turbinates, de-

flected septum, foreign bodies, specific adhesions, tumors of the nose or nasopharynx, polypi and acute infections.

There is only one well recognized method of treatment—making an opening through the obstruction and removing it, if possible. As the choanae at birth are very small, particularly in their vertical diameter, no large opening can be made. The opening should be made as soon as possible after the diagnosis has been established, in order that the patient may be able to eat and may not lose his sense of smell. Even though a permanent opening is not made, the proceeding is still capable of giving a not inconsiderable degree of temporary relief. Richardson considers it hazardous to operate before the patient is at least two years of age, and Syme holds that any opening made before the expiration of the second year will not be permanent; but I find myself unable to agree with either of them. It is my opinion that one may operate safely at any age, and that the opening, in most cases, will prove to be permanent.

This opening is more of a resection than a perforation, and it can be made by any cutting instrument of proper size, such as a trephine, drill, chisel, saw or biting forceps; but the secret of success lies in removing not only the occluding substance, but the posterior portion of the nasal septum as well. Dan McKenzie first did this in 1910. This tends to prevent recurrences, as a large cavity is produced which remains open permanently. Many operators report that they have succeeded in maintaining the opening by means of rubber tubes placed through the nose and changed frequently, for six months or more, or by running a strip of gauze through one nostril and out the other, retying and changing it daily (Brady[18]), but neither of these proceedings is necessary if the posterior portion of the nasal septum is removed.

At this point, I will report two cases of congenital occlusion that have recently fallen under my observation, cases illustrative of the points that I have just been making. Dorothy A., aged four months, referred by Dr. Martin Nordland, was born two and a half weeks prematurely, labor being so long that it was necessary to make use of forceps. She was a blue baby and breathing was established with considerable difficulty, the usual methods being of no avail. However, she began to breathe upon her mouth being held open forcibly. It was necessary to keep a nurse in constant attend-

ance to hold the mouth open, as the minute her vigilance was relaxed, the mouth closed, breathing ceased and cyanosis appeared. Respiration with the mouth open was very noisy, and the tongue had a tendency to cleave to the roof of the mouth continuously. It was found necessary to feed the child through a stomach tube. After the eighth day, she learned of herself to hold her mouth open and to breathe in that way, but cyanosis occurred at intervals, being relieved by spontaneous opening of the mouth on the part of the patient. There was a profuse yellow discharge from both nostrils, which poured out when the infant was held face down. The voice was clear from birth, chest examination was negative and no thymus enlargements were disclosed by the X-ray. A very small mass of adenoids could be felt in the nasopharynx. By means of examination with the nasopharyngoscope, a choanal occlusion was plainly to be seen. It was dimpled on the right side and solid on the left. It was impossible to pass a probe through either nostril. Attempts to blow air through the nostrils with a Politzer bag caused tears to bubble at the lower punctum of the eyelids. There was no deformity of the face, jaw, nasal septum or palate.

The left posterior nares was opened by means of a chisel, and the obstruction was found to be of solid bone, whereas the right posterior nares was obstructed by a substance that was part bone and part membrane. Both openings were thoroughly rasped with a frontal sinus rasp, and the new openings were curetted. The posterior portion of the septum was removed as a whole by means of a forcep inserted through the mouth. Neither tubes nor packs were left in the nose. The patient reacted well to the operation, but no improvement in the breathing was discerned until after the second day. Then I ran a silver Eustachian catheter through each nostril, well into the nasopharynx, at the same time holding the mouth closed. For the first time, the patient began to breathe through the nose, and she was able from then on to nurse normally, making the use of the stomach tube unnecessary. During the next two weeks, her breathing improved and she slept with her mouth closed. The yellow nasal discharge entirely disappeared. Six months later, examination disclosed that she was breathing normally.

The second case was operated upon by my former associate, Dr. E. H. Parker. At that time, the patient was seven-

teen years of age, and detailed a history of a right sided nasal obstruction with profuse yellow discharge and loss of the sense of smell on that side. Two unsuccessful nasal operations had been previously performed. Her condition was diagnosed by Dr. Parker as unilateral congenital occlusion of the choana. He made a permanent opening through the occlusion. I saw her seventeen years later, when she was thirty-four years of age, and the opening was still functioning satisfactorily.

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DISCUSSION.

Dr. Austin A. Hayden, Chicago: The paper of Dr. Phelps brings to us a very important subject that has been very little discussed. I take it that the reason why congenital strictures of the choanae are not more often seen is because they occur in very young children, and as Dr. Phelps has said, the nose breathing is completely obstructed and very frequently the "blue baby" dies without any sort of postmortem examination and without any nasopharyngeal finger examination after death, so that in many of these cases the condition is not seen.

Personally, I have never seen a case that I know of. The symptoms, however, of double occlusion as described, are typical. The symptoms of single occlusion, where one side of the nose is open and the other closed are less characteristic. Perhaps the first thing to be thought of is total obstruction of one side and the symptoms that follow. If the patient be put in a recumbent position and nose filled with sterile water, if after the nasal cavity fills, the water runs into the throat, that would be pathognomonic of obstruction of one side.

I think, from a practical viewpoint, perhaps the partial obstructions are much more important. I think, frequently we do not carefully note the size of the posterior opening. I do not doubt that many cases have been operated for nasal obstruction, tonsils and adenoid removed, and still the mouth breathing continued. If a careful examination and comparison of the size of the openings were made in these cases, very often it would disclose the fact, of which we have seen many examples, that either one or both posterior openings were small. If a prognosis is given at the time of operation that the mouth breathing will continue in spite of orthodontic measures performed in the nose and throat, a great deal of anxiety will be saved the operator.

In conclusion, I would say that the most important thing, it seems to me, for us to consider in this very important subject, is either the postmortem examination of these blue babies that die from respiratory failure, or a more careful examination of the nasopharynx; and in case of coming to operation for obstructed breathing, that a careful examination be made and comparison of the choanae on each side, and then see that this is carefully recorded as one of the essential causes of the persistence of mouth breathing.

Dr. B. N. Colver, Battle Creek, Mich.: I have seen only one case of complete congenital occlusion of the posterior choanae. Some six years ago, a child, two and a half years old, was brought to the clinic in this condition. I want to speak of the diagnostic points. Besides the introduction of the exploring finger posteriorly and a silver probe anteriorly, we tried closing one side of the nose and introducing the tip of a rubber bulb syringe into the other side and making slight air pressure. There was no passage of air. I thing that is a simple confirmatory diagnosis of complete occlusion.

In operating, a protecting finger (left index) is put into the nasopharynx, the right hand holding the gouge while the assistant taps on the chisel. In that way you avoid possible injury to the posterior pharyngeal wall and easily demark the button of bone to be removed.

Removal of the edge of the septum was brought out by L. E. White

of Boston, and appeared in the Laryngoscope, as a method of preventing closure. Up to that time, various devices had been used to prevent closure, but by the removal of that edge, none of these devices is necessary.

Dr. Joseph C. Beck, Chicago: I would like to ask whether an X-ray was made of these cases, because the X-ray does show some things.

Dr. Kenneth A. Phelps, Minneapolis, (closing): The test of blowing air into the nostril by means of a Politzer bag was performed in my cases, and I saw air bubbles appear at the punctum. This is a very good proof of complete nasal obstruction.

Another point should be mentioned—the sense of smell is absent in these cases but returns usually after the obstruction is relieved, as occurred in my second case.

As to the time of operation on a young baby; some men believe it is better to wait until the patient is two years old to render the operation more safe; others say no permanent opening can be made at an early age, and there is no use in operating. Personally, I feel operation should be done as soon as the diagnosis is made. Even if the opening is not permanent, it will allow the baby to eat without a stomach tube, and will help to save the sense of smell.

I believe Dan McKenzie was the first to remove the posterior portion of the septum in treating this condition. He did this in 1910.

In answer to the doctor's question concerning X-rays, no plates were made of the baby but were made of the adult.

SOME NASAL AND EAR DEFORMITIES CORRECTED BY NEW SURGICAL METHODS.

GEORGE B. JOBSON, M.D.

FRANKLIN, PA.

CORRECTION OF HUMP OR SADDLE BRIDGE NOSE. (SIMPLIFIED METHOD.)

In the July, 1925, number of The Archives of Otolaryngology, I published a brief outline of the operation which I shall describe, and referred to it as an original surgical procedure, for the reason that I had used it for the past six or more years, and was unable to find any reference to a similar method previous to my first description of the operation to a group of medical men while on board the S. S. Rotterdam on June 29, 1922. Since publishing the article, I have received communications from Dr. Albert E. Bulson, Jr., of Fort Wayne, Ind., and Dr. Robert H. Ivy of Philadelphia, Pa., who state that they have been performing nasal bridge operations by way of the nasal apex for some time. Dr. Bulson is of the opinion that Monk originated the idea of approaching the bridge from the tip and he kindly sent me a description of the method, which I note differs from mine in the direction of the skin incision, an important factor in favor of my procedure, as I will show later. Even though a limited number of operators may be correcting nasal bridge deformities through an incision of the nasal tip, I feel justified in bringing the idea to the attention of the profession more generally, because of its facility and safety; for the greatest deterrent to the correction of external nasal deformities is the difficulty of reaching the field without causing infection or visible scar formation. The removal of the enlargement in hump nose, or the placement of a cartilage or bone graft in saddle nose, is especially facilitated by the method to be described.

When a general anesthetic is administered, ether by insufflation is the method of choice. When possible, I prefer the use of local anesthesia with freshly prepared one percent

procain in physiologic sodium chlorid solution. The face is washed with warm water and soap, followed by the use of ether and alcohol. The hair is clipped from the interior of the nostrils, and each vestibule and the columella is cleansed with surgical solution of chlorinated soda. Nasal passages up to the vestibular junction are packed with half inch strips of sterile gauze, over which plugs of cotton wrung out of

Fig. 1. Skin incision at anterior part of columella.

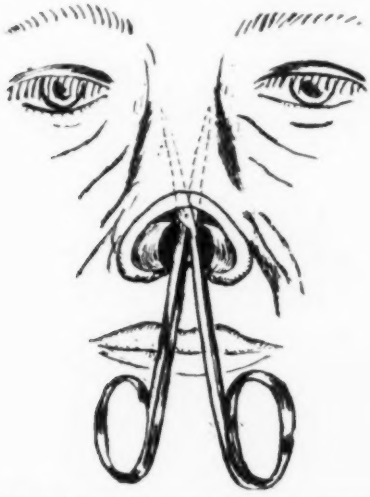

Fig. 2. Scissors inserted beneath flap and separating skin.

saline solution are placed, to prevent more effectually the oozing of blood into the throat.

A transverse incision is now made with a small, sharp scalpel through the skin of the anterior part of the columella, at its junction with the under surface of the nasal tip, and prolonged on each side along the anterior sharp border of the naris for about one-quarter inch. The skin is dissected from the tissues beneath, the flap is raised with a single or double tenaculum so as

not to crush it, a pair of small Mayo or similar scissors with blunt, but narrow points, are substituted for the scalpel, and the skin is easily and quickly separated over the tip, bridge, and sides of the nose if desired, by alternately opening and closing the scissors. This incision is sufficiently large to allow any instrument used in the operation to be freely manipulated, or a graft of correct size to be inserted. Such is not the case in the small vertical incision advocated by Monk. To quote from Kolle's Plastic and Cosmetic Surgery, under Monk's Method: "The difficulty the author finds with this method is,

Fig. 3. (Case 1.) Hump nose before operation.

that it is practically impossible to do good work with the scissors in this position."

For correction of hump nose, all loose tissue is freed and the periosteum is separated to each side with an elevator: then the hump is removed with a chisel, a small saw, or spoke shave, according to the will of the operator. I prefer specially designed rasps in different sizes. Bleeding is controlled by applying firm pressure from three to five minutes. The plugs are removed from the nostrils, stitches are taken with medium sized "dermal" suture, placing one suture in the center of the

Fig. 4. (Case 1.) Hump nose after operation.

columella, and one in the border of each anterior naris. The sutures should be placed close to the edges of the skin, and tied with barely sufficient tension to draw the skin together. Care should be exercised in approximating the skin edges accurately, as an uneven union will increase the tendency to scar formation. However, if a scar does form, it will not be observed unless the under surface of the nose is brought into view. The stitches should be removed on the third day. A pressure dressing, consisting of a piece of properly trimmed dental wax, is placed in warm water to soften, after which it is molded over the bridge and allowed to harden. Adhesive strips are used to attach this dressing in place, and it is left

Fig. 5. (Case 2.) Saddle nose before operation.

for forty-eight hours. Sterile petrolatum or albolene may be applied to the skin incision to prevent the formation of scabs.

The same technic may be followed in forming a bed for the insertion of ivory-cartilage, or bone-cartilage graft, for the correction of saddle bridge nose. A hump on the side of the bridge may be removed, or lateral depressions filled in with cartilage by this way. I believe this subcutaneous route offers a method which is superior to any other for the following reasons:

(1) If a scar forms, it is not observed under ordinary circumstances.

(2) Drainage is easily established, if required.

Fig. 6. (Case 2.) Saddle nose after operation.

(3) Approach to the field for all bridge operations is simplified.

(4) There is less danger of infection and less trauma than by the internal and nasal route, as it is necessary to raise the skin only over the bridge, and not from the side of the nose, in correcting hump or saddle bridge.

Depressed Nasal Apex with Low Columella.

This deformity may be corrected by making a transverse skin incision near the junction of the columella and upper lip. The skin covering the columella is next dissected free to the

Fig. 7. (Case 2.) Showing absence of scar.

nasal apex with a scalpel, and prolonged over the nasal tip and lower part of the bridge with a pair of slightly curved, blunt pointed scissors. The part of the columella which is freed from its coverings is now dissected with a short curved cut at the lip, concavity of which presents forward, so that the angle at the junction of lip and nose may not be too sharp, continuing the dissection to the apex of the nose without detaching the graft. A "dermal" suture armed with two

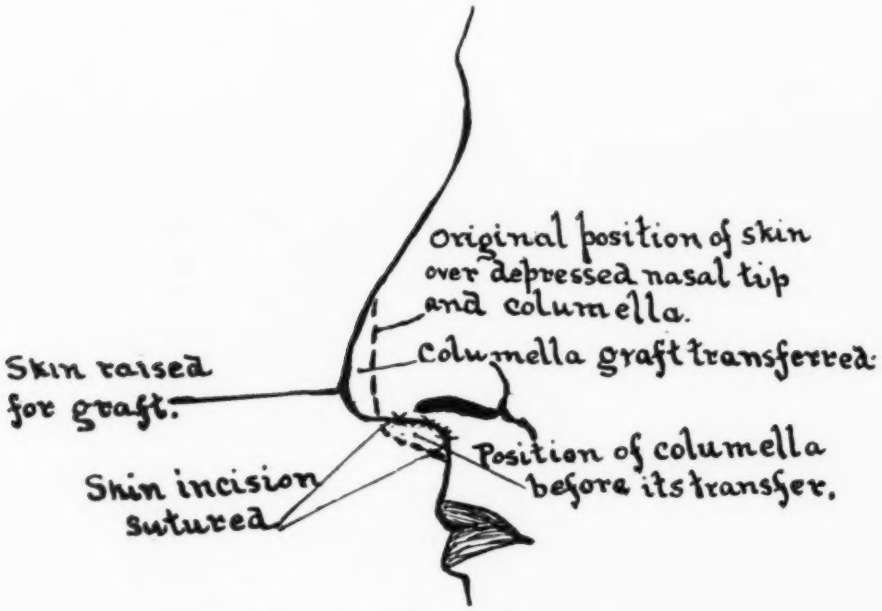
Original position of skin over depressed nasal tip and columella.
Columella graft transferred.
Skin raised for graft.
Position of columella before its transfer.
Skin incision sutured

Fig. 8. Depressed nasal apex with low columella.

half curved needles is inserted through the free end of the graft, the needles are then placed under the loosened skin over the nasal tip and pushed through the skin higher up. The graft is folded at its base and drawn into the bed prepared for it, by tightening the thread and tying lightly over a piece of small rubber tubing. The skin flap is carefully drawn back into place and sutured with small or medium size "dermal." A piece of dental wax is warmed, moulded over the bridge and held in place for two days with adhesive strips. All stitches are removed on the third day.

Collapsed Alae Nasi.

This condition, known also as pinched nose, results from a deformity of the alar cartilages, or else an atrophy of the muscular fibers supplying these structures. Great difficulty in breathing through the nose is often experienced in this condition. At times, an operator is chagrined when he discovers that a perfect operation on a supposed obstruction within the nose fails to correct, owing to the fact that the real trouble was collapsed alae, which were elevated during the examination and operation and consequently overlooked. A simple method of correcting a collapsed ala is to make a skin incision on the inside of the nostril, about one-quarter inch (0.6 cm.)

Fig 9. Right ala collapsed (Case 3.) Left ala corrected.

from its border, and extend it from as near the roof of the nose as possible to the lower third of the outer wall. The incision should be made in front of the depressed part, for the reason that the cartilage which is to be used is more easily inserted, better retained, and the stitching facilitated, than by an incision directly beneath the depression. Raise the skin

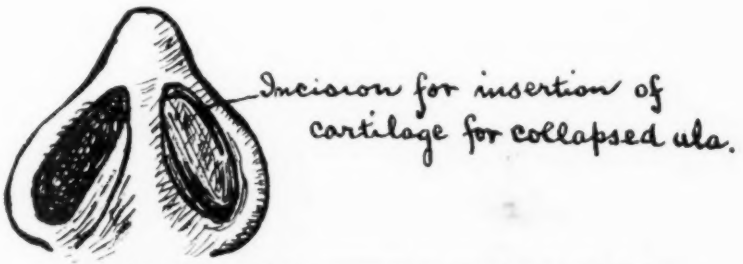

Fig. 10. Depressed ala corrected by insertion of cartilage.

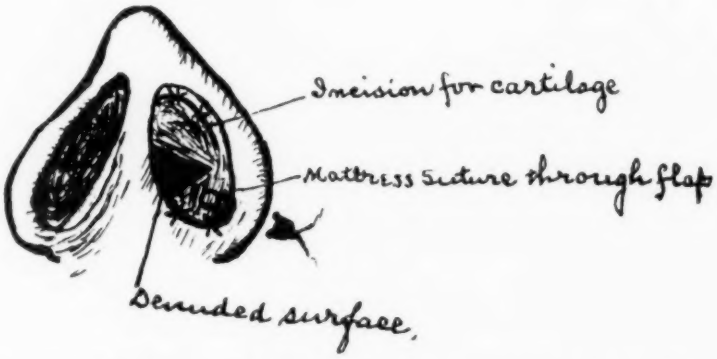

Fig. 11. Extreme case of collapsed ala corrected by Jobson-MacKenty method.

beneath the alar cartilage with an elevator or slightly curved scissors with blunt points sufficiently to correspond to the depressed area. Following the dissection of the skin, a piece of ivory or a thin piece of cartilage of sufficient size to elevate the ala is taken from the back of the ear, or costal region, and placed in position, and the skin stitched. A cylinder of gauze covered with tissue or sterile vaseline is inserted into the nostril and left for twenty-four hours. Stitches removed the third day. In extreme cases, it may be necessary to widen the floor of the nostril after the method of Dr. J. E. MacKenty. This

is done by dissecting a flap from the floor and septal side, extending the dissection backward beyond the ridge of bone which crosses the floor at the mucocutaneous junction. All unnecessary tissue is removed from the flap, leaving only cuticle and mucosa. The flap is swung outward, and its anterior border sutured to the line of the original incision. In addition, I use a mattress suture, by passing the needles from within out and through the flap and outer wall of the nostril, and tying on the outside. This fixes the flap firmly in its new bed.

Fig. 12. Depressed tip with (Case 4) low columella

Cauliflower Ear.

This ear deformity is characterized by thickening and distortion of the contour of the auricle. The condition is the result of hematoma auris, which becomes organized into cartilage and scar tissue. The treatment consists in making an incision in front of, and parallel to the rim of the ear. Next the skin is carefully dissected from its outer and inner surfaces, to the side of the head, if necessary.

Fig. 13. (Case 4.)

Fig. 14. (Case 5.) Cauliflower ear, saddle nose and thick lips resulting from inhuman treatment for 14 years.

Fig. 15. Cauliflower ear and rest of face corrected (Case 5) after 3 months' work.

The auricular skeleton is now sculptured into shape with scalpel and scissors. Absences in continuity of the rim may be corrected by stitching in pieces of cartilage with absorbable catgut. The skin flaps should have several cuts made in them for drainage . . . . be drawn over the modeled ear cartilage and their edges united in front of the rim again. Several mattress sutures may be passed through both layers of skin and cartilage to facilitate union. An even pressure should be maintained by filling all irregularities with cotton wet with salt solution, and allowing it to remain until healing takes place. A gauze pad and pressure bandage is applied.

DISCUSSION.

Dr. Joseph C. Beck, Chicago: The essayist said, "A plea for plastic surgery", and I thought probably he might have said, "A plea for a little less plastic surgery", because we find there is at the present time such a thing as an epidemic of this kind of work—in the correction of nasal deformities that really do not exist. That type of plastic surgery is one thing that is bringing this work into disrepute. We have a lot of gentlemen in this country, and especially since the World War, who have gone off at a tangent, allowing themselves to be dragged into operating on mental cases, which are giving them a great deal of trouble, and we see the result of their troubles considerably. I am sorry for these men, and I do all I can to minimize their trouble, because they are physicians, but really it is too bad that such a thing is going on and it should be changed.

The type of work that the essayist presented of course is not in that line. These were genuine cases of deformity, which required a great deal of study in order to get results that the patient and the doctor wish to obtain.

In regard to priority in the matter referred to, long ago someone described an operation of an incision from the junction of the columella and the upper lip, but that is not the point; the main proposition in plastic surgery is to consider the principles, the fundamentals underlying the repair. The objection from that point of view to any operation through the tip of the nose is the possibility of secondary perichondritis, which may result from too free dissection of the dermal part of the nose; that is not loose but is intimately connected, and when you begin to dissect extensively you are in danger of perichondritis. This means the shrivelling and shrinking and atrophy of some of the cartilages, or a productive inflammation of the cartilages. That is the reason I choose the intravestibular route, or through the eyebrow. That part of the vestibule may be isolated by firmly packing off the nasal cavity and working within the vestibule. This can be done with perfect safety and no scar need be feared.

The choice of incision is invariably in the vestibule in the correction of depressions or humps.

In regard to the cauliflower ear, the majority of ear men who are doing this work are disappointed in the results from plastic reconstruction—they are very poor. I believe the time is coming when a properly made prothesis will be the thing so far as the correction of external ear deformities are concerned.

In regard to transplanting to correct defects, if you use a septal cartilage you will note it will be absorbed. Cartilage obtained from the pinna may be used as stiffening. It will hold for some time, but if you have collapsed alae that do not respond to that stiffening, then make a perforation of the septum. A properly healed perforation will prevent a falling in of the alae. Of course we do not want to make a perforation of the septum intentionally, but that is the only way the alae can be kept open at times.

I would make a plea for plastic surgery before this body—major plastics in deformities where parts are absent. Such major plastic work entails the use of tissues from the forehead, arm, etc. Such surgery will bring this plastic work the dignity that it deserves.

Dr. Sidney Israel, Houston, Texas: Dr. Beck discussed the development of the incision and his preference for the intravestibular route. I, personally, prefer the intravestibular route for two main reasons.

First: No sutures are required after the operation is completed.

Second: There is no external scar produced as a result of the incision.

In the external incision, or an incision through the columella, whether it be transverse or vertical, a sufficient working space is not always obtainable, particularly in certain types of deformity of the nose, i. e., the lateral deviation. In my opinion, this is the most difficult deformity to correct, and requires ofttimes the maximum of working space on each side of the bony framework of the nose, for unless this bony framework is corrected, no satisfactory result can be expected.

In the incision through the columella, the attachment of the skin over the depression in the alae is likely to be disturbed, and when this attachment is disturbed, an abnormal appearance of the nose takes place, which is noticeable in the end result.

In the event of an infection through an external incision, whether it be in the columella or elsewhere, prolonged drainage would more than likely add to the scar that would naturally result. I feel that the intravestibular incision gives greater working space on the lateral side of the nasal bones, and in the event of an infection, drainage can take place with no visible scar possible, due to the location of the incision.

In the correction of the dorsal hump deformity, it is necessary to arrange the contour of the nasal bones after the hump has been removed, otherwise there will be a shelf like appearance of the dorsum of the nose, which is quite objectionable.

The after care in any type of nasal deformity is important, and a splint should be used. I have tried the many types of splints for this purpose, including modeling compounds, but have chosen the most excellent splint, recommended by Dr. Cohen to be the best, i. e., the copper splint. This splint is easily sterilized and made to fit the individual case, and readily maintained in place by adhesive plaster.

We have had no infection whatever through the intravestibular incision in any of our cases, and I feel that by this method of approach we are, at least, not doing anything likely to produce a scar. I must state my position again—that any technic wherein a scar is likely to be produced in the correction of a deformity, if it can be avoided, is fundamentally lacking, and in no part of the body is this more important than in the correction of deformities of the nose.

In ear deformities, of the cauliflower type, or prize fighter's type, we use a similar technic to Dr. Jobson's, and feel that a most essential point is contact of raw surfaces, after the blood clot has been removed. By contact, we lessen the chances of infection and hasten recovery.

Some form of modeling compound, or plaster, is needed to keep the flaps intact, but the flaps should be perforated so as to facilitate drainage.

I have had several cases of atresia of the nostrils in children; one in a child injured during delivery, with atresia of one nostril. This condition is most difficult to correct, and it is my opinion that, after the plastic operation, the use of metal dilators in graduated sizes, over an indefinite period, is necessary to bring about the perfect end result.

THE DEVELOPMENT OF THE SINUSES AND THE INFLUENCE OF INFECTION UPON THEIR DEVELOPMENT.

THOMAS E. CARMODY, M.D.

DENVER, COLO.

While it is not the object of this paper to give a review of literature, or conduct a class in history of the work on the paranasal sinuses, it is quite necessary to make a few preliminary observations. Many of you have read the books and papers which I will only mention in passing, as milestones, and many others, and to those who have not, they are recommended as highly desirable literature.

We are prone to consider that we of the last decade were the discoverers of sinuses as real pathologic entities, and while I do not mean to disparage the recent excellent work, we must remember that the sinuses were discovered by Vesalius in 1535, and that the present work of investigation was born about 53 years ago with the paper of F. Steiners in 1872. Following this came Zukerhandl in 1878; J. Bland Sutton, in 1884; M. H. Cryer, 1892; A. Onodi, 1893; G. Killian, 1895; Herbert Tilly, 1896; A. Logan Turner, 1901; J. W. Murphy, 1901; J. M. Ingersoll, 1906; J. P. Schaffer, 1910; W. B. Davis, 1914; L. W. Dean, 1917, not to mention the various books and papers which deal more especially with surgery of the sinuses.

These papers and books, with the exception of the last two, deal almost entirely with the anatomy of the adult sinuses and their variations from normal. Furthermore, it was largely a study of anatomic specimen, mostly dry and fully developed. While much of the work done has proven of great value, some conclusions have been found to be incorrect in the light of later investigations. Many have been incorrectly quoted, which makes for confusion. This, however, is not necessarily intentional.

While pathologic conditions of the sinuses are credited as the primary focus or the exciting cause of many diseased organs far removed, and while they must share this odium

with infected tonsils, teeth, etc., that is not sufficient reason for ignoring their existence or forgetting that some of them are present at birth, while others depend, we believe, on the normal functioning of the above for their progression.

We have attempted to outline what is normal and differentiate the pathology in the developing child. To this end, studies (roentgenographic) were made of children at term, immediately after birth. First of the chest, to note the expansion of the lungs. These were made by using a bedside unit, which is placed in the delivery room, exposures being made at once, and at intervals of 5, 10 and 15 minutes and 24 hours, and again before the child left the hospital. This work was done by Dr. W. W. Wasson under the Selem Winter Foundation, and is still being carried on with slight changes. The first was begun in 1922, and in the latter part of the same year I became associated, and the work of the ear, nose and throat was thereafter included in the examination.

This work now includes routine examination of the head, chest and abdomen, both clinical and roentgenographic, noting the agreement or disagreement of findings, with the addition of a complete reexamination every three months, or at more frequent intervals if the mother or attending physician notes any variation from normal.

These children come from all classes and include not only the ordinary clinic family, but the middle class and a few that may be termed wealthy. Also, we find many from normal parents, and a few from families of whom one or both parents are tuberculous.

It was early noted, that some of the older children (as this work is only in its fourth year, some of the children have not been through the whole course, but were taken as a rule because of some suspected pathology) four years and older, did not show normal development of the frontal sinuses and mastoids, and while the latter had been worked out by Witmark, nothing had been done on the former. Soon after this, however, Shea suggested an analogy with Witmark, based on operative findings. As many of these children never come to operation, and may not even present symptoms, we feel that we are possibly doing something from the standpoint of prophalaxis, and have noted changes due to infection, malnutrition or other dietary disorder, as observed by Dean.

In the time allotted, we cannot explain pathologic conditions of the chest, so often arising; neither can we discuss surgery, except to note that such was necessary.

It is notable that many operations for tonsils and adenoids are performed on children whose main pathology is in the sinuses by general practitioners, general surgeons, and we are sorry to say also rhinologists, because a complete examination has not been made. Many of these cases will later require sinus drainage, either therapeutic suction or surgical, although some may clear up after normal ventilation is established by removal of adenoid plug from the nasopharnyx.

The slides will show, first, that good roentgenograns can be made of sinuses even if only partially developed, although we have heard the statement made that children should be anesthetized. Second, slight changes may be noted due to infection or other irritation. Third, abnormal development was found due to infection, most frequently in the maxillary sinuses. Fourth, that this infection not only affects the mucous membrane lining the sinus, but also the periosteum, and mainly the osteogentic layer of this membrane.

We must disagree with Shea that children show atrophic condition most frequently, as in many the membrane is hypertrophic.

SLIDES.

1.	Davis	95	days.	Embryo, coronal sec.
2.	Davis	120	days.	Horizontal sec.
3.	Davis	at term.		
4.	Author.....	7	mos.	
5.	Author.....	10	mos.	
6.	Author.....	14	mos.	Seeley
7.	Author.....	14	mos.	
8.	Author.....	14	mos.	David.
9.	Author.....	20	mos.	Taylor.
10.	Author.....	22	mos.	Warren Williams
11.	Author.....	2	yrs.	Raymond Williams.
12.	Author.....	2	yrs.	Sutton.
13.	Author.....	2	yrs.	Glantz.
14.	Author.....	11	mos.	Geo. Bell, 2 sec.
15.	Author.....	25	mos.	Geo. Bell, clear.
16.	Author.....	26	mos.	Faegert.
17.	Author.....	28	mos.	Max. Sinus slight rarefaction; beginning in the sphenoid.
18.	Author.....	3	yrs.	Hall.
19.	Author.....	3	yrs.	Peck.
20.	Author.....	5	yrs.	Wilma Jean Dugan Frontal.
21.	Author.....	5	yrs.	Emily Hinds.
22.	Author.....	5	yrs.	Tilley.
23.	Davis	7	yrs.	Adenoid Plug.
24.	Davis	6	yrs.	Sphenoid.

25.	Davis	6	yrs.	Frontal.
26.	Davis	6	yrs.	Sphenoid.
27.	Davis	6½	yrs.	Frontals not developed.
28.	Davis.....	8	yrs.	Findlay, acute max., frontals developed.
29.	Davis.....	9	yrs.	John Smith, Chronic max., frontals not developed.
30.	Davis	11	yrs.	Lyon King, frontals developed.
31.	Davis	11	yrs.	Arthur King, frontals not developed.

DISCUSSION.

Dr. J. J. Shea, Memphis, Tenn.: The ethmoidal cells are present at birth. They develop until puberty is well established, and reflect in their growth the influences of the infections they have suffered. The sphenoid sinus is generally a distinct bony cell at birth, but has no clinical significance. From the age of three, the sphenoid sinus grows rapidly, so that it reaches its adult size at the age of ten. The frontal sinus is formed by the migration of an anterior ethmoid cell into the frontal bone, and as soon as the cell enters the frontal bone, it becomes the frontal sinus. At birth and during the early years of life, the ethmoid cells are sharply divided into an anterior and a posterior group. The sphenoethmoid labyrinth will assume the size and shape its component bones will allow, and the presence or shape of the frontal sinus is governed by the migration of the anterior ethmoid cells. The growth of the sphenoethmoid labyrinth should be proportionately pneumatization and capsulation. This growth is dependent upon the absence of infection, for the sinuses reveal in their growth and development the infections they have suffered. If the infection at any time during the developmental period of the sinuses is severe or persistently mild, it will retard or stop the natural growth and development, with the production of a type of sinus in the adult known as the persistent infantile sphenoethmoid labyrinth. In the persistent infantile sphenoethmoid labyrinth, the pneumatization is subnormal and the capsule hypertrophied, as shown by the roentgenogram. The characteristic persistent infantile sphenoethmoid labyrinth in the adult has few anterior cells, and these are small with hard, thick walls, which do not migrate sufficiently to form a frontal sinus; or if they do form a frontal sinus, it is only rudimentary. The posterior cells are likewise few with hard, thick walls, but are larger than the average posterior cell, though their aggregate volume is less. The sphenoid sinus is small, and its walls are thick and firm. The membrane lining these cells is thin and not capable of normal secretory powers. Atypically placed cells are very common. The persistent infantile sinuses are prone to atrophic diseases and osteitis, while the normal sinuses are capable of recovering from severe purulent inflammations. In the latter group, chronic inflammations may produce hypertrophic changes and polyps. The cystic type of ethmoids can occur only after the sinuses have been normally developed, for we do not meet any cystic changes in the persistent infantile type. In fact, the cystic changes are really pathologic degenerations and not developmental changes. When infections have arrested the development of the sinuses, this is not always permanent, for by reestablishing the normal ventilation and drainage of the undeveloped sinuses through surgery, they will begin to grow again and in many instances regain the lost development in a comparatively short

time. In fact, it has been my experience that the sinuses must show new growth before the pathology can be considered corrected. The teeth are influenced by the arrest of the development of the antra, but we have not had any evidence of injury to the further development of the teeth due to surgical interference.

Dr. Harry W. Lyman, St. Louis: I have two slides that Dr. Carmody desired me to show, and in this case evidently the development of the sinuses was not arrested by early infection. This is a frontal view, and this is the lateral. You can see that the sinus extends around on the side.

Dr. Anna B. Leffler, Los Angeles, Cal.: I would like to submit the report of an autopsy from the contagious ward of the Los Angeles General Hospital.

I appreciate that this is not a contribution directly in line with Dr. Carmody's research. However, the authentic record of frontal sinusitis in a five year old child is of value for the proceedings of the Academy.

This child died of meningitis following whooping cough. The autopsy showed all nasal sinuses were filled with pus, except the right antrum. The frontal cells were about the size of a pea and were packed with thick pus.

The meningeal infection had occurred through a dihiscence of the lateral wall of the sphenoid, and had travelled backward along the sheath of the fifth nerve.

Dr. John A. Cavanaugh, Chicago. I have been taking X-rays before and after injecting the sphenoid sinuses with barium suspended in malted milk. My work in this line is not complete. In these X-ray pictures, it is a surprise to find that areas outlined as the sphenoid sometimes prove to be incorrect. The cases I have been working on are adults.

Dr. Ernest F. Risdon, Toronto, Canada: A lantern slide demonstration is rather difficult to discuss, but I gather from Doctor Carmody's remarks, that he wishes to emphasize the importance of the relationship between the teeth and the antrum. One point that was not brought out was, "How can one know?" The Doctor made the statement that a number of these teeth are close to the antrum, but do not puncture it. The method he would have told you, without doubt, is the method we all use, and is the use of the stereoradiogram. Dental X-ray will lead you into endless trouble. From the standpoint of the antrum being high or low, it has been maintained that intranasal drainage of the antrum that comes below the level of the floor of the nose will not be effectual. That statement, gentlemen, was made from the standpoint of the investigator rather than of the man who is treating these cases, because I am sure you have found that drainage of the nose from that standpoint is successful.

If the drainage is into the mouth, one rather infers that the antrum is not involved, or that the infection is not due to the teeth. The men who are practicing oral surgery, I believe, state that as high as 50 or even 70 per cent of infected antra are of dental origin. The reason, I suppose, is that as the rhinologists see it almost entirely from the standpoint of systemic condition, the oral surgeons see only cases that are

referred because of tooth infection, consequently believes the incidence of infection is greater from that standpoint.

Another point is that investigation with the X-ray, as Doctor Carmody has shown, sometimes shows that the antrum frequently comes forward and almost meets in the middle line. If the anterior teeth do become infected quite early and it is necessary to destroy the nerve, of course it would penetrate the antrum at that position, but it does not follow, that simply because a front tooth is infected, it does not puncture the antrum. We have had in our clinic a case where the central or lateral was perhaps one inch away from the anterior border of the antrum, but on operation we found a sinus through the bone and emptying into the antrum. We hardly expected that, because the anterior plate of the alveolar process is so thin.

I would like to compliment Doctor Carmody on his slides, and I am sure I feel as he does, that not enough attention is given to this matter, and that simply because teeth are infected, it does not follow that the antrum is infected.

Dr. Thomas E. Carmody, Denver, (closing): I have not much to add, except to state that in my paper I attempted to parallel the work of Whitmaak, which Dr. Shea has mentioned, in the subject of the undeveloped mastoid, my paper dealing with the undeveloped sinuses. I also wish to state that in this work we have been X-raying these sinuses about every three months, or oftener, provided there seems to be any trouble noted by the mother or family physician.

I was glad that Dr. Leffler reported her case because we know that many cases that have died of infectious diseases show pus in the region of the mastoid and sinuses.

Dr. Cavanaugh spoke of the injection of the sinuses. I cannot help but ask the question, whether he really knew he injected the sphenoid. I do not know whether I could tell or not. There is a possibility that we might miss the sphenoid sometimes.

Doctor Risdon brings out some important points which I did not mention—of course I overlooked a number of things, speaking as I did. One is the stereoradiogram. It will tell you absolutely whether the root is in the maxillary sinus or not. Sometimes we do not believe it is, but we want to have the best possible interpretation of the plain film. We should not, of course, extract teeth on slight evidence.

Doctor Risdon brought out another important point, that of intranasal drainage; and there comes to my mind another point, that is, that in many cases where infection has already made an opening into the maxillary sinus, we extract the teeth, intranasal drainage is at once instituted, and the opening will close; but if it is allowed to go for some time, it may require further operation in order to close it. I have seen two cases within the last few days, where the rhinologist had attempted to close the oral opening without nasal drainage, and of course both of them broke open.

Doctor Risdon spoke of the large percentage of infection from the teeth. Of course it depends upon where we see our cases, and further, what pathology we are looking for.

One point I want to leave with you, is to look out for the first permanent molar. Too many rhinologists overlook that when examining

the condition of the throat and nose, and allow the first permanent molar to decay and abscess, and possibly infect the maxillary sinus. I think if we were more careful in examing the mouths to see if there are decayed teeth, we would help these little patients very much. When we examine these first permanent molars, we frequently find they have decayed and should be either removed or taken care of at the time tonsils and adenoids are removed. We overlook them because many of us do not realize that they are permanent molars. The mother always thinks they are temporary teeth, because the child has not lost any teeth. But we have development of that process in the temporary teeth, and further development in the permanent teeth.

HYPERTHYROIDISM—ITS SIGNIFICANCE IN OTOLARYNGOLOGY.

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Perhaps there is no disease regarding the etiology and management of which a greater diversity of opinion exists, or about which a larger literature has accumulated, than is the case with hyperthyroidism The condition is here denominated "hyperthyroidism," although as will be indicated below, this term is considered a misnomer by those who do not consider that the syndrome so designated is due to hyperactivity of the thyroid gland.

As to etiology of the condition, I think that it can definitely be stated that it is unknown, most of the theories which have been proposed being concerned with the actively exciting factors of certain symptoms of the disease rather than with its primary cause. The great diversity of opinion regarding the primary cause of hyperthyroidism is indicated by the list of theories propounded by different authors, as given in Bram's recent monograph;—the bulbar theory, the intoxication theory, the kinetic theory, the thyroid insufficiency theory, the neurogenic theory, the toxic neurogenic theory, the dysthyroidism theory, the pluriglandular theory, the vagotonia or sympatheticotonia theory, and respectively the neuroendocrin theory, the sympathetic, the thymus, adrenal, parathyroid, pituitary, gonad theories. There obviously is not time for me to enter into any discussion of the content of these theories, but in any case they have no bearing upon the purpose of this address, which has to do with the syndrome of hyperthyroidism once it has become established.

The general course of hyperthyroidism, in contradistinction to the variety of causes which are attributed to it is strikingly uniform. Gradually or rapidly increasing symptoms of general nervousness, tachycardia, tremor, disturbances of

menstruation, and evidences of increasing vascularity of the thyroid gland, are the outstanding signs. Barker gives the following concise picture: "We can scarcely do better than to urge the practitioner to think of the possibility of hyperthyroidism in every case in which he meets with (1)*tachycardia*, (2) *rapid emaciation*, (3)*excessive sweating*, (4) persistent *watery diarrhea* without apparent cause, (5) *neurasthenic state*, (6) outspoken *lymphocytosis*, (7) one or more of the *eye signs*, and (8) *fine tremor*." It will be noted that in neither of the above lists of symptoms of the disease is mention made of exophthalmos, and that Barker mentions the eye signs among the last of the symptoms in his list. This seems to me to be an important note, for often a case of hyperthyroidism is missed because eye signs are not present, the term exophthalmic goiter having led too many of us to consider that that symptom is a "*sine qua non*" of the condition.

The acute type of hyperthyroidism needs no discussion. Its clinical course is outspoken and unmistakable. The subacute types are those which may require the finest clinical judgment for the establishment of the differential diagnosis. Among the conditions which might most readily be mistaken for hyperthyroidism are neurasthenia, neurocirculatory asthenia, early stages of pulmonary tuberculosis, paroxysmal tachycardia, and cases of myxedema, even though that is definitely a condition of hypothyroidism.

As for the general management of the disease, here again we find in general a uniformity of opinion that the one desideratum is the lessening of the thyroid secretion, but divergence of opinion as to the methods by which that end is to be attained, whether entirely by medical management, which includes the removal of whatever existing or promoting causes of the syndrome are present—such as focal infections, sources of emotional or mental strain, excessive physical strain, and so forth—or actual diminution of the amount of the secreting gland by the X-ray or by a surgical operation. In the Cleveland Clinic, the last cited is the method of choice, for the reason that it is possible to control exactly the extent to which the activity of the gland is lessened, as is impossible when the X-rays are used. Other reasons for this choice which are at variance with the belief of many advocates of the X-ray cannot be given in detail here.

A word should be added regarding the incidence of hyperthyroidism and regarding certain characteristics of hyperthyroidism in children, as they have an immediate bearing upon the special problem of this discussion. First as to incidence. We are so accustomed to consider that goiter and many diseases of the thyroid gland are in the main confined to certain fairly well limited goiter districts, that we may overlook the fact that hyperthyroidism has no such limited distribution. Those of us, therefore, who are outside of the goiter districts should bear the possibility of this condition well in mind, in view of the dangers to be considered later, which may arise from ill advised procedures upon patients with hyperthyroidism. The same caution applies also in the case of children. We have been apt to consider that hyperthyroidism is a disease of the later years of adolescence and of early adult life. Its incidence in young children has been generally supposed to be so rare, that it is hardly to be considered. In the Cleveland Clinic, however, forty cases of hyperthyroidism have been seen in children under fourteen years of age, and various groups of cases in children have been reported in the literature at varying ages under fourteen years, one being only nine months old. In our series, the ages varied from five and one-half to fourteen years, and in one case it appeared from the history that the disease was initiated when the child was but three years of age. Although this is not a high incidence as compared with the cases of hyperthyroidism encountered in later years, it is sufficiently high to warn us that we should be especially on our guard when children come to us who present a syndrome of symptoms which suggests the presence of hyperthyroidism. In general, however, the diagnosis should rarely be mistaken in such cases; the symptoms are usually outspoken in children and the characteristics of the onset are fairly uniform. Parents usually first notice increasing nervousness and irritability, which is followed by a rapid enlargement of the gland, tremor and in many instances—fifty per cent in the Cleveland Clinic series—by exophthalmos.

A word regarding the pathology of the disease may be of interest, particularly since it can be confined to the one statement that there is no specific pathology. There is no known change in the thyroid gland itself, or in the organism as a

whole, which is pathognomonic of hyperthyroidism. The fact that there is no specific pathology is one of the reasons why the etiology and exact character of the disease is so uncertain.

As to the establishment of the diagnosis in uncertain cases, various tests have been suggested which may be made in adult cases but are of little or no value in establishing the diagnosis in children. Among these tests are basal metabolism estimations, the Goetsch adrenalin test, the Braun quinin test, and examination of the blood. The warmest supporters of any of these methods, however, themselves emphasize the fact that these tests are of minor value as compared with the anamnesis and the clinical findings.

It is of special importance that the physician be not misled by the size of the goiter. In some cases, also, in which there are no external evidences of thyroid enlargement, a goiter may be present which is extending down into the chest. These intrathoracic goiters often bring to the otolaryngologist some of his most serious problems.

The role of infection in the etiology of hyperthyroidism and goiter as indicated above is uncertain. Its role in the course of hyperthyroidism on the other hand appears to be definitely established. It may be stated here, however, that although it would appear that, in general, hyperthyroidism is not caused by infections, so many instances in which hyperthyroidism has immediately followed an attack of influenza have been noted, that this fact may be of some etiologic significance. Aside from this one instance, however, the role of infections seems to be at present an exacerbation of the hyperactivity of the thyroid gland. This hyperactivity of the thyroid gland, in turn, produces an increased susceptibility to the infections, so that a vicious circle is established.

The role of infections in cases of hyperthyroidism in children is of especial importance to the otolaryngologist. In eight of our series, there was a history of an antecedent infection; in one, the onset of the disease appeared to be directly related to an attack of whooping cough; in another, exophthalmos developed three weeks after an attack of scarlet fever. Cases are reported in the literature by Klein in which hyperthyroidism developed immediately after tonsillectomy, and one case is reported by Wheeler in which exophthalmos followed an attack of varicella and mastoiditis; in this case

the hyperthyroidism was associated with status lymphaticus. Although the percentages are small, nevertheless the close association of the development of hyperthyroidism with the previous infection is certainly significant.

Those who believe that hyperthyroidism has a neurogenic origin cite cases in which the syndrome has immediately followed shock, fright or great emotional strain. One of our cases in the children's series immediately followed an unfortunate experience in a dentist's chair. The child went home in a state of great emotional excitement, with greatly accelerated heart action, and within a week, though kept in bed, she exhibited the characteristics of a typical hyperthyroidism syndrome, including early exophthalmos. Crotti reported a case operated upon at Kocher's Clinic, in which the symptoms of hyperthyroidism appeared within 48 hours after the San Francisco earthquake. I believe, however, that in cases which follow exanthemata or surgical procedures, such as tonsillectomy, an unrecognized state of hyperthyroidism already existed, which was aggravated by the infection, operation, or other exciting factor.

That any focus of infection should be cleared up is unquestionable. But when it should be removed is again a problem, the solution of which is not easy. In cases of an intense hyperthyroidism, the removal of infected tonsils, the clearing out of sinuses, or the extraction of teeth may be sufficient to kill the patient. In children, in particular, in the extraordinarily hypersensitive state produced by the hyperactivity of the thyroid gland, mistimed operative procedures are peculiarly dangerous. In children, also, the presence of a thymus gland often associated with hyperthyroidism may add materially to the operative risk.

The prime problem which confronts the otolaryngologist, then, in connection with hyperthyroidism, is on what basis to judge what shall be the primary procedure, that is, whether or not the patient shall be referred to the surgeon or the roentgenologist for primary treatment of the hyperthyroidism, or whether the infection shall receive the primary treatment. The necessity for recognizing the condition in patients who come to him either of their own volition, or sent by physicians who have not recognized the signs of hyperthyroidism, is obvious, and the otolaryngologist should be informed regarding the relative merits of radiation and of surgery in the

treatment of the disease, and of the methods employed by the internist, in order that his judgment may be based upon a thorough knowledge of the most efficient prevailing practice. For myself, although radiation therapy has many adherents, whose statistics appear to justify their claims, I believe that the surgical removal of the gland is the only method whereby the hyperactivity can be directly and permanently controlled.

In addition, even in an extremely mild case of hyperthyroidism, in which it may appear both to the otolaryngologist and to the referring physician that removal of the focus of infection may be a sufficient measure to control the hyperactivity of the thyroid, the former should inform himself thoroughly by a physical examination and with the aid of the roentgenogram as to the relation of the gland to the trachea. It is of prime importance that the otolaryngologist know whether or not the trachea is in any degree distorted or compressed by an intrathoracic enlargement or other pathologic condition within the chest, which may increase risk of the operation. In general, I might state that it is my own conviction, and I believe that this judgment is shared by the surgeons of the Cleveland Clinic, that any otolaryngologic operation should, if possible, be deferred until the operation has been directed to the cure of the hyperthyroidism itself. The tragic sequelae of tonsillectomies performed upon patients with hyperthyroidism illustrate dramatically the extent to which the effects of each are intensified by the other. I believe that during operations certain deaths have occurred which have been due to unrecognized hyperthyroidism, or to some mechanical condition such as pressure upon or distention of the trachea by a goiter. In some cases the patient does not recover promptly from the anesthetic; nausea and vomiting persist, the pulse rate mounts to an alarming degree, to 150 or 160, there is intense restlessness and delirium may develop. It is because of this dangerous sequence of events, which almost invariably follows a tonsillectomy performed in the presence of hyperthyroidism, that we believe that whenever possible primary attention should be directed to the cure of the hyperthyroidism.

In some cases, the only operation indicated may be the ligation of the superior thyroid arteries. This is especially true in children, in whom, as Dr. Dinsmore has informed me,

the improvement following ligation is often far more pronounced than that which is seen in adults. But, as a rule, the control of the hyperthyroidism is obtained only after the thyroidectomy is performed, two or three months after the ligation. It should be emphasized that a tonsillectomy or other operative procedure should not follow the operation upon the thyroid within less than from four to six months. The otolaryngologist should also bear in mind that the surgical operation is but one step in the cure of hyperthyroidism. A complete hygienic nursery regimen is required for a period, the length of which is in direct relation to the length of the period during which the disease was developing. Hay has recently called attention to the importance of a thoroughgoing detoxication in cases of hyperthyroidism, by means of daily enemas of cool water, sweats and a carefully chosen diet in order to maintain as low a nitrogen index as possible. As toxic conditions are characterized by a high nitrogen index, this would seem of especial importance in the type of case which comes to the otolaryngologist, in which the toxins of an infection are added to the toxins engendered by the hyperactive thyroid gland. In view of our present understanding regarding disturbances of the interrelation of the endocrine glands in this disease, it is necessary that a period for the reestablishment of the normal relation be allowed. Since the effects of an infection upon the organism are, in many cases, so nearly identical with the effect of hyperthyroidism, it follows that in these cases even more than the usual care should be given to the control of the hyperactive heart, to restoration and conservation of the function of the kidney, and to the control of environmental conditions—that is, to the assurance that excitement and worry are eliminated. This is peculiarly the case with the patient in whom the hyperthyroidism is complicated by the presence of an infectious process.

Even when every possible precaution has been taken, a period of adequate rest should follow a tonsillectomy. This is of the greatest importance if no operation for the thyroid has been deemed necessary, but even when a preliminary ligation or thyroidectomy has been performed and an adequate interval has been allowed—even then the patient should be required to rest in bed for at least one week. After operations of a more serious nature, an even longer period of rest in bed should be insisted upon, and it should be the practice of the

otolaryngologist also to insist upon a prolonged period of rest away from school or from work, and when the patient goes home and the care of the otolaryngologist is ended, he should be referred to his own physician for a protracted period of watchful care and guidance.

In certain cases, such as an acute suppurative mastoiditis, when the operation cannot be delayed, the resources of the specialist may be taxed to the utmost. For the control of such cases, he should have at his command every possible restorative and conserving measure. Acute mastoiditis in the presence of hyperthyroidism is a very menacing situation. In such a case, the specialist should protect himself by insisting upon the presence of the surgeon or of the patient's own physician. In case an operation under local anesthesia is contemplated, the fact that thyroid activity is increased by adrenalin shows the importance of omitting the use of adrenalin if it is at all possible to get along without it. More sedative measures should be employed before and after operation than is the case in the usual uncomplicated operation. That is, the preoperative distress of mind and the postoperative distress of body should be reduced to the very minimum. As Crile has shown, every nervous reaction and even the slightest degree of pain has its effect upon the organs of the kinetic system, among which the thyroid plays a major role. The less pain, the less nervous excitement, the more rest, the more sleep, by so much the more will the general welfare of the patient be promoted and the danger of increased hyperthyroidism be lessened. The heart should be fortified by digitalis if there is the slightest indication of myocardial weakness. To use Crile's dictum, the restorative and conservative measures should be used "in advance of the emergency." Because of the increased heart action, more bleeding than usual occurs during certain operations, and there is greater danger of secondary hemorrhage; therefore, before operation, the blood of the patient should always be grouped and provision made so that a blood transfusion can be done without delay should it be necessary.

It will be noted that I have made no attempt to distinguish between hyperthyroidism and the socalled toxic adenomata. If such a distinction is of value, it does not pertain to this discussion, for as far as the otolaryngologist is concerned, his management of the case should be as outlined.

The specialist should therefore be familiar with the symptoms and causes of hyperthyroidism, and with the most efficient methods for its treatment for the following reasons:

1. The relation of infection to hyperthyroidism is not definitely known, and the removal of a focal infection is not a cure for the hyperthyroidism.

2. Under ordinary conditions, and especially in children, the surgical removal of a focal infection should be deferred until the hyperthyroidism has been controlled by surgical treatment.

3. A sudden death during an operation upon the throat which is often attributed to the anesthetic or to cardiac failure, may be due to the presence of unrecognized hyperthyroidism.

4. Throughout the entire conduct of a case of hyperthyroidism, unusual preoperative and postoperative precautions are essential.

5. When it is imperative that an operation be performed in the presence of hyperthyroidism, the presence of the internist at the operation and his cooperation during the period of convalescence are of utmost importance.

DISCUSSION.

HENRY B. HITZ, Milwaukee: The subject, "Hyperthyroidism—Its Significance in Oto-Laryngology", is one which is constantly met with in laryngologic practice and I think deserves more consideration than it has received in the past. Experience has shown very conclusively the connection between some vocal paralyses, acute throat infections, and, particularly, chronic septic conditions of the tonsils with thyroid disease; and it has been *so* recognized by the surgeons, that now cases are constantly being referred to the laryngologist for careful examination, prior to operations upon the thyroid. For many years past, I have urged upon my surgical friends the importance of throat examinations prior to the goitre operation. By this time, most of them have become aware of the necessity for it, from their own sad experiences.

One of the altogether too common complications, fortunately less marked today than a few years ago, is that of "partial recurrent paralysis" following thyroidectomy; and particularly serious is the form "bilateral abductor paralysis", with its concomitant results. It has been assumed by many surgeons in the past, that this was due to injury done to the recurrent laryngeal nerves, resulting from operation. In some instances this was unquestionably too true. Examination prior to operation, however, shows that preexisting unilateral abductor paralysis in goitre cases is not uncommon, and the knowledge of this is of great importance

to the surgeon, who in the subsequent operation can exercise every possible precaution to avoid injury to the uninvolved side.

I have had under my observation in the past two years six cases of bilateral abductor paralysis, four of whom are leading tubal existences because of the inability to correct the condition, notwithstanding the excellent operation of ventriculocordectomy of Jackson, or the recurrent nerve anastomosis of Jackson and Frazier.

Regarding the infective side of this subject, I can best express myself by reading to you a brief resume of the case record of a girl of fourteen, with acute exophthalmic goitre, with final practically complete cure, resulting from the clearing out of large badly infected tonsils and adenoids, three months after ligation of both superior thyroids seemed to have made but little impression, and when her condition did not seem to permit of thyroidectomy.

Miss Olive K., age 12. Entered Milwaukee Hospital Dec. 16, 1921. Family history negative.

History of disease: Parents noted increasing nervousness and developing goitre and exophthalmos preceding April, for which local treatment was instituted.

Physical examination—main points: Marked exophthalmos, large soft goitre, cardiac hypertrophy, pronounced tonsil hypertrophy, tachycardia, normal temperature, pulse averaging 140—highest point 168.

Treatment: Absolute rest, digitalis and occasional opiate. On Dec. 22, ligation, under local anesthetic, Rt. superior thyroid. On Jan. 3, 1922, the left superior was ligated. (Dr. Evans.) Discharged Jan. 8.

Returned to hospital March 21, held until the 29th, when she was sent home again with a notation, "Patient not in condition for thyroidectomy" by Dr. Evans. The chart shows a general improvement in tachycardia—highest point pulse 150, average 136.

Returned again to hospital May 8, 1922. Some general improvement noted. Dr. Evans advised tonsillectomy before thyroidectomy. Operated under ether, May 10, elapsed time 12 minutes. Nature of operation—tonsillectomy (dissection method) and adenectomy.

Pathologic Diagnosis: Chronic follicular tonsillitis (tonsils contained much cheesy material). Large mass of adenoids.

Discharged in fair condition May 15—pulse ranging 120 to 130. Seen six months later—complete disappearance of exophthalmos. Pulse, respiration and temperature normal. Apparent complete subsidence of thyroid mass. Seen October 15, 1925—apparently normal—well nourished girl of 16, slightly overweight—eyes large—beef eye type—common to other members of family. Pulse rate average about 80.

The house surgeon who watched this case stated that the basal metabolic rate at the time she was at the worst was the highest he had ever observed, viz. 137 plus.

Much more might be said upon this subject, did the time allotted to me permit. Suffice it to say that I am in complete harmony with Dr. Waugh in his desire for closer approximation of the surgeon and the laryngologist in the observation, care and treatment of thyroid conditions.

Dr. John N. Coghlan, Portland, Oregon: I apologize for my appearance again on the platform in the role of Calamity Jane. I also want to congratulate Dr. Waugh on his paper. Coming as it does from

perhaps the largest goiter clinic in the United States, it has a real message for the nose and throat specialist.

Three months ago, a patient treated by a family physician was sent to me to have tonsils out. She was a young lady, just through High School, who had tonsils and large adenoids. I advised her to have the tonsils out under general anesthesia because of the large adenoid. The parents of this girl were friends of 25 years standing and had all the confidence in the world that what I would do would be right. I assured them that I would be very careful, that I would inject the tonsil with novocain and adrenalin to be sure I did a good job. This I did, and was assured by the nurse that the novocain and adrenalin were all right. I had taken out one tonsil and was changing the mouth gag to get at the other one, when the patient went into the most violent spasms I have ever seen and immediately collapsed. We did everything we could think of to restore her, but after about an hour's work she was pronounced dead. A most searching autopsy was made, and the pathologist found it was a pronounced case of status lymphaticus. I had begun to think there was no such thing, because in 25 years I had never seen a case or had a death.

I give this as a warning to laryngologists not to be too precipitate in operating without examination for glandular disease.

A NEW PROCEDURE FOR THE POSTOPERATIVE CARE OF LARYNGOSTOMY.

HARRY L. BAUM, M.D.

DENVER, COLO.

In presenting my method, I make no especial claim for priority, as some such procedure may have been advocated previously without my knowledge. However, so far as I have been able to discover, this mode of treatment overcomes one of the most trying obstacles to the successful management of these cases, and does so without danger or discomfort to the patient. I have found no previous mention of a similar method in the literature.

The object of the device presented is to prevent contraction of the laryngostomy wound, and to maintain a good exposure over a long period of time—as long a period as may be necessary for the proper dilatation of the stenosis. This it does in a very simple manner, without requiring the use of any packing in the wound following operation and without pain or discomfort during the period of healing.

The device consists of the addition of a post to the ordinary laryngostomy tube or, preferably, to the Tucker tube, which is the one I use in the illustration. This post is calculated to fit the laryngostomy incision snugly, and is so designed that the upper or saddle end extends from the rubber tube inside the larynx to the skin, this saddle being wide enough to give the necessary "spread" to the upper end of the incision. The tube itself does the same for the lower end, leaving the edges free between these points. Thus, the epithelium grows in and rapidly covers the raw edges of the wound without the necessity, in most instances, of drawing the skin and mucosa together.

The laryngostomy is done in the ordinary manner and the tube is inserted immediately. No packing is used except the ordinary dressing externally, and this is changed as often as necessitated by the secretions. Should the raw surfaces show

Fig. 1. Tucker laryngostomy tube with post attached.

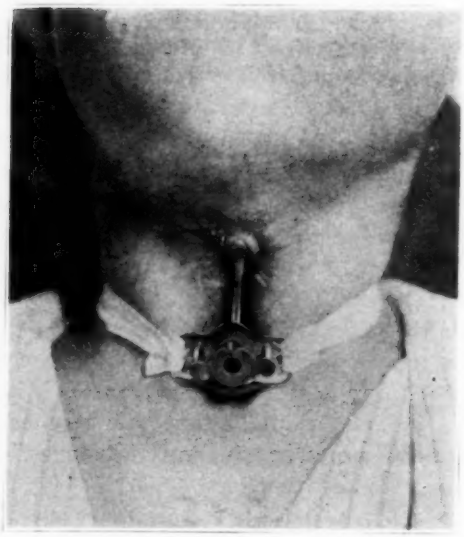


Fig. 2. Showing apparatus in place.

Fig. 3. A typical laryngostomy incision after wearing apparatus six months.

Fig. 4. Showing the extent of exposure.

a tendency to too free bleeding the first twenty-four hours, a light gauze pack may be introduced along the sides of the post for its control. This will not usually be found necessary, as a well fitting rubber dilating tube will prevent much leakage into the trachea, even though surface bleeding may be considerable.

Subsequent dressings consist in the simple changing of the tube, which causes very little discomfort. The skin edges are sensitive for a week or two, but this quickly subsides as healing progresses. Healing is rapid and with a minimum of granulation, although there is some thickening and growth of granulation tissue at the upper end of the post around the saddle.

The post takes the place of the wire which on the Tucker tube is for the purpose of tying the thread for anchorage of the dilating tube. I secure excellent anchorage by bringing the thread around the tracheotomy tube, tying it on top, where the knot is out of the way and cannot irritate the skin.

The presence of the post does not materially increase the difficulty of slipping the dilating tube down over the "up and down" portion of the apparatus when it is in place. In fact, the excellent exposure obtained in this way and maintained by the continued use of the apparatus makes the changing of tubes and dressing of the case a very quick and simple procedure. The patients quickly become accustomed to the presence of the post and do not object to it in the least.

A GENERAL UTILITY OPHTHALMIC LAMP.

JAMES M. SHIELDS, M.D.

DENVER, COLO.

The lamp I am presenting is the result of the development of an idea originally suggested to me by Dr. Melville Black's method of oblique illumination. He had placed an automobile spot light lamp in a skiascopy chimney, and in front of the aperture in the chimney had attached a plus 20 D. lens, thus producing parallel rays. For concentration of this light upon a given spot, he had employed a condensing lens held in the hand in the usual manner. The evident usefulness of this simple arrangement suggested to me the possibility of the development of a general utility ophthalmic lamp, which might be adaptable to a great variety of uses in the hands of the ophthalmologist. After considerable experiment, I have succeeded in producing a lamp which I find highly satisfactory in my own practice, and I am submitting it for your approval.

In my practice I use the lamp for the following purposes:

1. Oblique illumination, either with white or with red free light.
2. Ophthalmoscopy, using either white or red free light.
3. Retinoscopy, either with the ordinary illumination or with the small aperture suggested by Dr. Edward Jackson for centric retinoscopy, or with red free light in patients showing a certain degree of photophobia.
4. The projection of histologic eye sections.
5. As an operating light.

The lamp, itself, consists of a ventilated hood, so designed with interior baffling and asbestos sheathing, as to give perfect ventilation to the globe without light leakage, thus reducing the heating of the hood to the minimum and preventing condensation of moisture on the lens, with resultant fogging. Attached to the hood is a holder designed to take the ordinary plus 20 D. trial case lens, which, at this distance, pro-

Fig. 1.

Fig. 2

duces parallel rays. Anterior to this holder, is a specially devised double holder for the purpose of receiving the red ray filter when in use, and also accommodating the microscopic slide when it is desired to use the lamp as a projector. The front holder is also designed to accommodate the ordinary trial case lens, and in addition has an attachment with 2 mm. aperture, which can be swung into place behind the lens for use in centric retinoscopy. The lamp is readily adjustable on its stand to suit any position of the patient, or it can be mounted on a handle and used as a hand lamp. The transformer has been placed in the base of the stand, where it is not in the way and is out of sight. These features can be readily noted on the accompanying illustrations.

For oblique illumination, with a plus 20 D. lens in the first holder producing parallel rays, the addition of a plus 20 or a plus 10 D. lens in the anterior holder produces a steady, sharp point of focus, very useful in the removal of foreign bodies from the cornea and showing beautifully pathologic changes in the anterior segment. With dilated pupil, lens changes may be readily seen. By inserting the red ray filter, increased vascularity in any part of the anterior segment is brought out in a very striking manner. The choice of lenses for the anterior holder in oblique illumination is practically unlimited, as a great variety is available in the trial case and the choice is dependent only upon the focal distance required.

For ophthalmoscopic examination of small areas of the eyeground the lamp is ideal, giving a source of light which is intense and which can be made red free by the insertion of the filter.

In retinoscopic examinations, I make use of centric retinoscopy as suggested by Dr. Edward Jackson; using the 2 mm. aperture attached to the anterior holder for this purpose. The lamp is equally adaptable to ordinary retinoscopy, however, using preferably a plus 5 D. lens in the anterior holder. With some photophobic patients, I find the red free light a great convenience for retinoscopy.

For projection of histologic eye sections, any degree of magnification may be secured from about 4 x. to about 70 x. With this range, one may demonstrate very clearly the relationship of different parts of histologic sections in a manner not possible with the naked eye or with the microscope. The

latter, even with low power, includes too small an area in its field for this purpose.

When using the lamp as an operating light, a plus 10 D. lens will be found more practical in the anterior holder, although any selection may be used at the discretion of the operator. For this purpose the lamp is especially satisfactory, giving an even, brilliant spot at any reasonable distance, in any position and of practically any size.

The particular features of the lamp that I hope will appeal to you, are its extreme adaptability to practically any procedure in our routine work, its adjustability, the red free light feature, the very brilliant, clear, steady light produced, its simplicity, and the fact that every oculist has in his equipment a full complement of lenses for use in the lamp for every conceivable purpose.

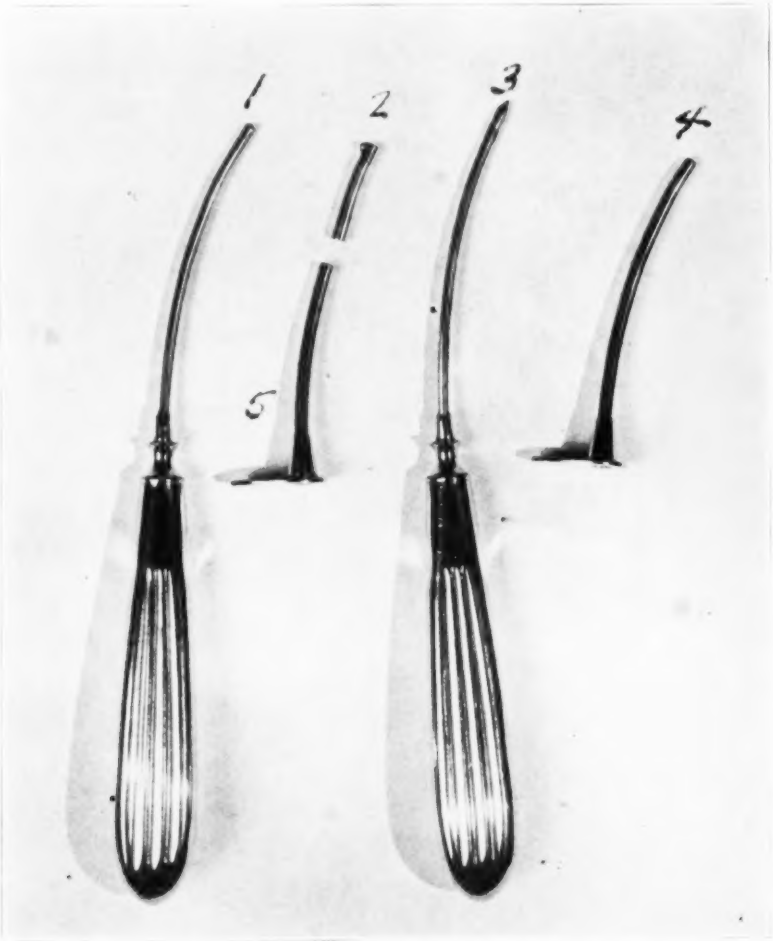
MAXILLARY SINUS TROCAR WITH RETAINING CANULA.

LaVerne B. Spake, M.D.

KANSAS CITY, KANSAS.

Dr. L. B. Spake showed a maxillary sinus trocar with retaining canula for use in children, for drainage and aeration.

Made by Erschell Davis Co., Kansas City, Mo.



1. Blunt pointed trocar. 2. Retaining canula. 3. Sharp pointed perforator. 4. Canula for adults. 5. For inserting retaining canula in sinus.

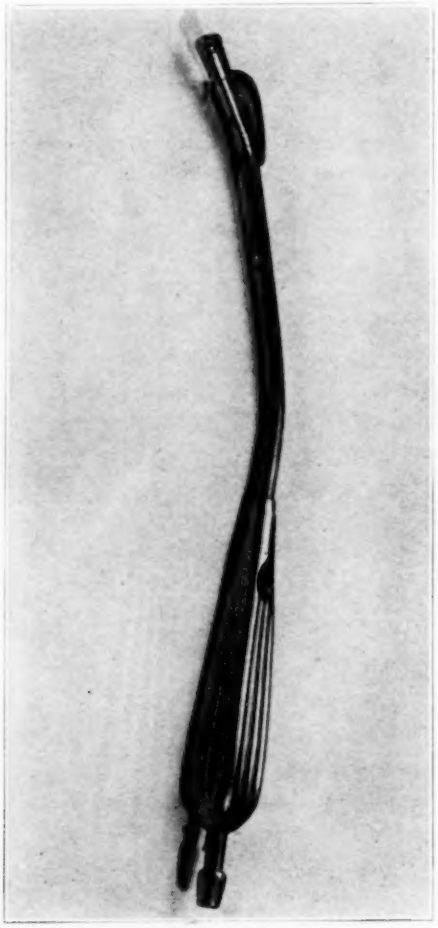
SUCTION TUBE WITH TONGUE DEPRESSOR.

J. A. Fulton, M.D., and L. B. Spake, M.D.

KANSAS CITY, KANSAS.

Drs. Fulton and Spake showed a suction tube (Sorensen) with tongue depressor.

Made by Erschell Davis Co., Kansas City, Mo.



Suction tube (Sorensen) with tongue depressor.

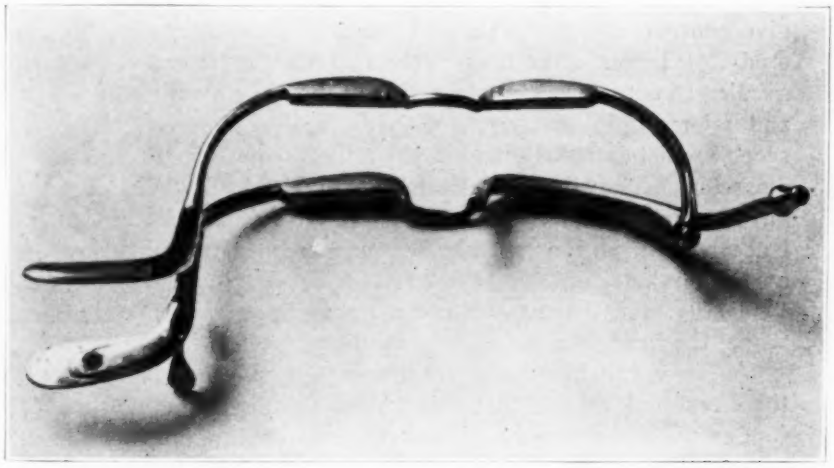
MOUTH GAG.

J. A. Fulton, M.D., and L. B. Spake, M.D.

KANSAS CITY, KANSAS.

Drs. J. A. Fulton and L. B. Spake showed a mouth gag. The lips are increased in size and leaded, ether attachment from left side, "Lockite" to right.

Made by Erschell Davis Co., Kansas City, Mo.



Mouth gag.

COMBINED FOCAL ILLUMINATOR, OPHTHALMOSCOPE AND RETINOSCOPE.

CHAS. A. BAHN, M.D.*

NEW ORLEANS, LA.

Will you please criticize this instrument, which I have been using and improving during four years? It is a combination focal illuminator, ophthalmoscope and retinoscope, which can be attached to an ordinary light socket.

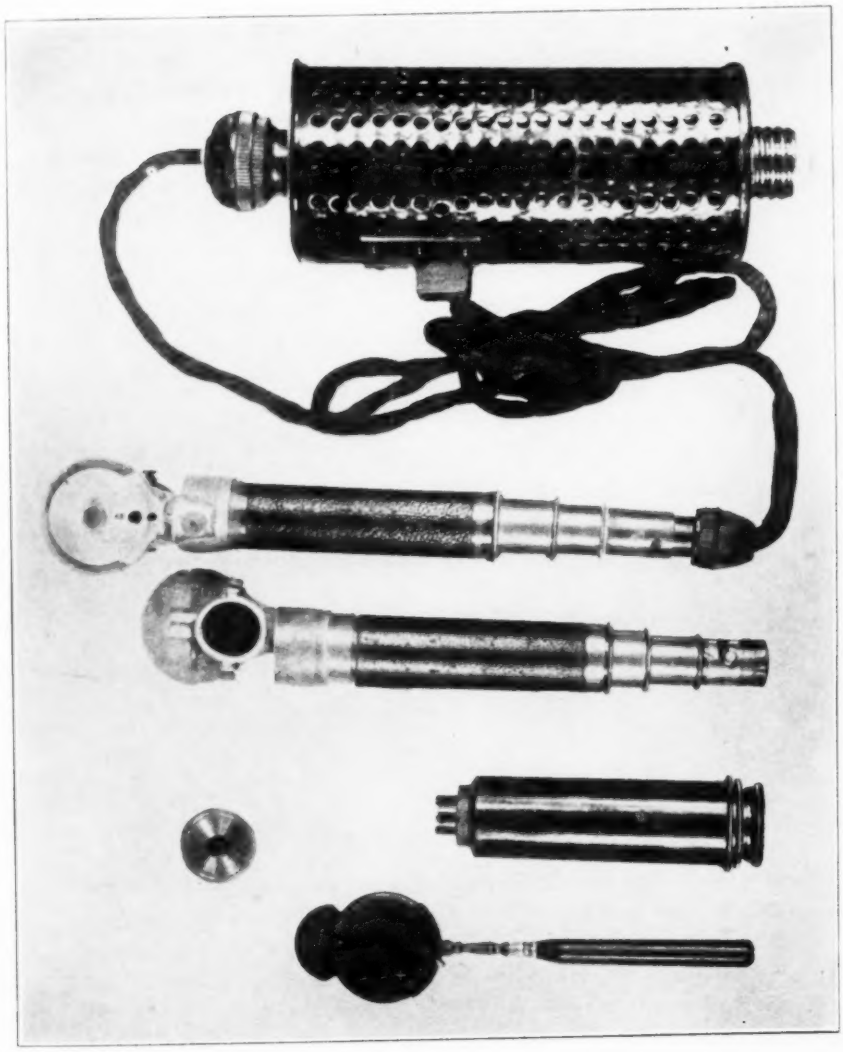
The focal illuminator with five light intensities, affords divergent, convergent, parallel and focal illumination for study of larger structures such as facial asymmetry, etc., smaller structures such as the lids, lashes, conjunctivae, etc., and microscopic structures, with the aid of a small microscope, which affords magnifications of from ten to thirty diameters. The slit light effect is easily obtained. With it, I have been able to see many of the practical details which can be seen with the larger, rather cumbersome, instruments now on the market.

To use the instrument as an ophthalmoscope or retinoscope, the head is attached as a cap.

As an ophthalmoscope, the instrument may be used with the direct and indirect methods. With the direct method, the fundus may be studied with convergent, focal, parallel or divergent light, each of which have respective advantages. A practically reflexless view of the fundus may be obtained by slightly turning the head on its axis. A small red absorbing disc permits of red free examination.

With the indirect method, one can study the fundus excellently illuminated. The Recos discs enable one to use lenses varying from plus 25.0s to minus 25.0s, which may be altered in units of one, five, ten, and twenty diopters without passing through intermediate lenses. The lens cover may be rémoved by simply unscrewing a small thumb screw, enabling one to lift out the Recos discs easily. The lenses can be

*Prof. Ophthalmology, Postgraduate Dept., Tulane University.



cleaned by a circular hand movement, which means that all the lenses can be cleaned with one movement rather than individually, as is now necessary.

The retinoscopic estimation of refraction can be made in several different ways by extending the tubes slightly; one can thus verify the results obtained by the plane mirror method with those by the concave mirror method. The small lens battery enables one to estimate refraction quickly. It is held in the hand of the observer close to the patient's face, the lenses being turned as required.

With this instrument, I believe that we have in a simple, practical form a combination of focal illumination, the corneal microscope, various kinds of ophthalmoscopes and retinoscopes, which I hope will be of practical value and will compose in a great measure all the equipment generally necessary to examine eyes objectively.

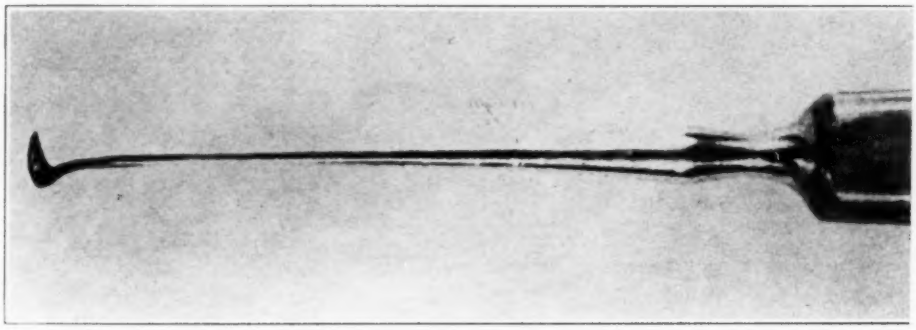
There are several unique attachments which I have in mind but which are not practically proven. As soon as the instrument is fool proof and sufficiently simplified, it will be available for public use, and I hope at a cost of about one-tenth that of corresponding instruments now in use.

CORNEAL FOREIGN BODY INSTRUMENT.

CHAS. A. BAHN, M.D.*

NEW ORLEANS, LA.

In the removal of corneal foreign bodies, it is important that trauma be minimized, that every particle of the foreign substance be removed, and that only clean instruments be used. These requirements are met in the instrument shown, which is made of rustless steel to facilitate quick chemical sterilization. It has a sharp cutting point on one side, a more rounded sharp edge for scraping on the other, and a broader



surface between for levering. One has, therefore, a miniature knife, scraper and lever combined in a single instrument. Accidental perforation is minimized, because the cutting surfaces are used only from the side and not from the point.

*Prof. Ophthalmology, Postgraduate Dept., Tulane University.

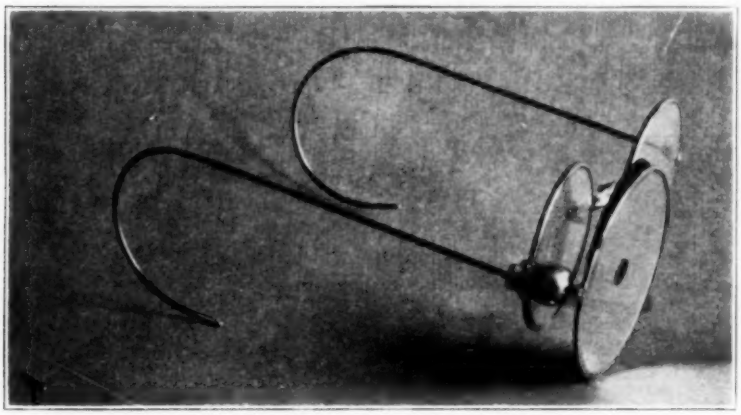
LEMERE-NETTLE REFLECTOR.

H. B. LEMERE, M.D.

OMAHA, NEBRASKA.

1. Light weight, as the mirror is small (2″ with ¼″ aperture) and has no metal back.

2. It can, if desired, be fitted with a ring carrier so that lenses of different focus can be brought behind the aperture; this model has +1.50, +2.25 and +3.00, the latter being very useful for the ear examination.



The advantages of this instrument are:

3. The mirror can be swung around to the temple when not in use.

The spectacles can be lensless and used over the wearer's regular glasses, or they may have his distance or near correction as desired, or as advised by his ophthalmologist.

The freedom from weight and from the binding of any headband is, to most laryngologists, a most welcome relief.

The manufacturers are planning to make this mirror so that it can be attached to any spectacle frame by means of a simple screw.

TRANSACTIONS

OF THE

THIRTIETH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

NECROLOGY
MINUTES
MEMBERSHIP ROLL
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FIVE YEAR INDEX

DEATHS

Dr. Samuel Ellsworth Allen, Cincinnati, Ohio.

Dr. Hugo W. Aufmwasser, Covington, Ky.

Dr. Frank E. Auten, Belleville, Ill.

Dr. F. E. Cheney, Boston, Mass.

Dr. Claude A. Freligh, Springfield, Mass.

Dr. William Allen Hollis, Hartford City, Ind.

Dr. Frank Jacobi, Toledo, Ohio.

Dr. Thomas F. Keller, Toledo, Ohio.

Dr. Isaac A. Lederman, Louisville, Ky.

Dr. Louis F. Love, Philadelphia, Pa.

Dr. Alexander Covington Magruder, Colorado Springs, Colo.

Dr. James McElroy, Meridian, Miss.

Dr. George H. Savage, Memphis, Tenn.

Dr. William Koenig Spiece, Chicago, Ill.

Dr. Henry Burt Stevens, Boston, Mass.

Dr. Robert Stevenson, Cincinnati, Ohio.

Dr. Roscoe Conkling Wilson, Ithaca, N. Y.

MINUTES.

October 20, 1925, 10 A. M.

The Thirtieth Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology was held at the Sherman Hotel, Chicago. The first Joint Scientific Session was called to order at ten o'clock by the President, Dr. Horace Newhart, Minneapolis.

DR. LUTHER C. PETER, Secretary, read a telegram of greeting from Dr. W. D. Haggard, President of the American Medical Association.

THE PRESIDENT: We are very fortunate in having as our guest of honor this year, a man who has the distinction of belonging to the second generation of a family of oto-laryngologists. Dr. Nager's father before him was a prominent otologist of Europe, and Dr. Nager stands preeminent in his country today as a man of wide experience in research and as a clinician. In fact, his laboratories have been the envy of all Americans who have visited him during the past few years. It gives me great pleasure to introduce to you Professor F. R. Nager of Zurich, Switzerland.

PROFESSOR F. R. NAGER: Mr. President and Officers of the Academy: Only a few words to thank you for your kind invitation to attend your meeting, for the very kind words of your President, and your signs of good will. I have been only a short time in your imposing country, and further am embarrassed by the defects of my English, but I hope to be able to show you what we have been doing the last few years, especially as the result of the training of my two former teachers, Dr. Siebenmann of Bâle, and Dr. Bezold of Munich. I will do the best I can.

October 20, 1925, 8 P. M.

The first Business Session was held Tuesday evening, October 20th, at eight o'clock, the President, Dr. Horace Newhart, presiding.

On motion of Dr. Arthur J. Bedell, duly seconded, the

reading of the minutes was dispensed with, as they had already been published in the Transactions.

It was moved by Dr. M. A. Goldstein that the Council make it possible that the slides shown by Professor Nager be made available to the members of the Academy who wish to study more minutely the research problem which he has presented—that this question be referred to the Council for consideration. Motion seconded and carried.

The Secretary, Dr. Luther C. Peter, read the following report, which on motion of Dr. E. C. Ellett, duly seconded, was accepted.

The Secretary's Report.

Mr. President and Fellows of the Academy: The membership of the Academy now totals 1,477. During the year, death has claimed 21 members. There are 3 resignations, and to offset these inroads upon the membership, there are 122 applicants for membership.

The general policies of the Academy are continuing along lines of expansion which have been in evidence since its inception, thirty years ago. During the last year, the American Board of Examiners in Oto-Laryngology began its activities. Oto-Laryngology, in the qualifications required of candidates for membership in the Academy, is now on the same basis as that of Ophthalmology. Other societies in these branches of medicine are conforming to the same standards. It is gratifying to remind our membership that this advance in Oto-Laryngology, as well as in Ophthalmology, is due largely to the Academy's influence.

By a vote of the membership two years ago, the program has been so arranged as to enable those who are interested in all the specialties of eye, ear, nose, and throat to attend all the scientific sessions. This plan has been on trial for two years, and it remains for the Fellows to decide this year whether the plan, which has worked to the satisfaction of so many of the members, shall continue in force in future arrangements.

A second change from former customs is that of the special evening business session. The business of the organization has increased during recent years, because of an increased membership, and especially because of the larger field of

activities to which the Academy has been dedicated. Long business sessions held before our regular scientific session has robbed the program of much valuable time, and has unnecessarily prolonged both joint sessions. The holding of a special business session, at which the business of the Academy may receive due consideration, without encroachment upon the scientific sessions, should commend itself to the entire membership.

Each year complaints of members who have failed to receive their Transactions reach the Secretary's office in increasing numbers. Some of these failures to receive the Transactions are due to incorrect address, others to unpaid dues, and a few copies are lost in the mail. During the last year, a questionnaire was forwarded to each member, asking for a complete report of the correct spelling of the name, the address of the member, the specialties which he practices, and the date of his entrance into the Academy. Up to the present time, not over 50% of these replies have reached the Secretary's office. With a fuller cooperation of the membership in supplying this information, and in responding promptly to the Treasurer's bills, which are sent to each member January 1st, your officers hope to reduce this annoyance to a minimum. Every effort is made by the Editor of the Transactions, the Treasurer, and the Secretary, when these complaints are filed, to adjust matters to the satisfaction of members, and in keeping with our By-Laws. Fuller cooperation, however, on the part of delinquent members will greatly aid in the adjustment.

Respectfully submitted,

LUTHER C. PETER,
SECRETARY.

NATIONAL BOARD OF EXAMINERS IN OTO-LARYNGOLOGY.

There is no special report to make, except the fact that there have been 355 men examined and certificated. At this meeting we examined 132, and the report will be sent out within the next few days. Dr. Wherry, the secretary, has done very efficient work, and the other committees from the other societies have worked very well with us, so that we feel the certification of the oto-laryngologists is on a par with the certification of the ophthalmologists, and we hope to have practically all who are entitled to certificates examined and certificated within the next five years.

THOMAS E. CARMODY.

On motion, duly seconded, this report was accepted.

Report of the Treasurer.

Cash on hand from 1924	$ 7,247.90
Received from dues, application fees, interest on bonds, interest on savings account, exhibits, etc.	21,443.89
Total Receipts	$28,691.79
Disbursements	9,389.62
Balance	$19,302.17
Purchased bonds	$14,005.40
Balance	$ 5,296.77
Previous bonds	$63,543.67
Grand Total	$68,840.44

Moved by Dr. Walter R. Parker that the report be received and that an Auditing Committee be appointed to audit the books of the Treasurer. Motion seconded and carried. The President appointed as an Auditing Committee Drs. A. J. Bedell and Harry S. Gradle.

American Board for Ophthalmic Examinations.

It is just ten years since the first meeting for organization of this body was held in Chicago, in connection with the meeting of the American Academy of Ophthalmology and Otolaryngology, which had chosen representatives to join with those of the American Ophthalmological Society and of the Section on Ophthalmology of the American Medical Association for this purpose. The certificate of the Board has now been awarded to 501 applicants. During the past year, 51 certificates have been awarded by the Board.

During the nine years since the first examination, held at Memphis, December, 1916, there has been a steady increase in the interest in these examinations, and a steady and gratifying improvement in the preparation of the applicants who appeared to take them. This progress has been shown this year in the two examinations, held in Philadelphia in May

and in Chicago yesterday. The organization of the National Board of Examiners in Oto-Laryngology has manifestly quickened the interest in such examinations, and the great interest that has developed in graduate courses in the two specialties bears eloquent testimony to the timeliness and importance of the movement in which this Academy has taken an important and honorable part.

Respectfully submitted on behalf of the representatives of the Academy.

EDWARD JACKSON.

On motion of DR. E. C. ELLETT, duly seconded, this report was accepted.

Moved by DR. W. R. PARKER that the reports of other committees be postponed until the business session Wednesday morning. Motion seconded and carried. The business session adjourned.

October 21, 1925.

The Business Session was called to order at nine-fifty.

The NATIONAL BUREAU OF MEDICAL RESEARCH had no report to make. On motion of DR. T. E. CARMODY, duly seconded, this Bureau was discontinued.

REPORT OF THE DELEGATE TO THE INTERNATIONAL CONGRESS OF ENGLISH SPEAKING OPHTHALMOLOGISTS.

I do not believe a formal report is necessary, further than to say that the meeting was a very interesting and profitable one for all of us. We were royally entertained. The character of the work was highly satisfactory from a scientific standpoint, and before the meeting adjourned, a committee was appointed the purpose of which was to look forward to a similar meeting at some future time.

LEE M. FRANCIS.

On motion of DR. LUTHER C. PETER, duly seconded, this report was accepted.

SECTION ON INSTRUCTION.

The Section on Instruction has no specificity to report—the work is open and announcments have been sent out to the members. The Committee is instructed to continue, and trusts the membership of the Academy will continue to give

the committee the same support, and the benefit of their suggestions and criticisms that they have in the past. The Committee is merely trying to put before the Academy what the membership seems to want. It is not our endeavor to force any of our ideas. We are somewhat handicapped by not receiving as many suggestions as we would like, and if the membership at large will please feel free to send in suggestions, both constructive and destructive, your committee will appreciate it greatly.

The work of the Section on Instruction will start Thursday night with an open meeting, and will continue through Friday and Saturday. The feature that we had at one time, namely, the Round Table, is to be started again this year, and from the registration it would seem as though that feature was enjoyed by the membership at large. Those who register for the Round Table are requested to write out such questions as they may desire to have discussed and hand them to the presiding officer at the individual table, either at the beginning of the luncheon or between this time and that. The round tables are in small groups, and therefore it is hoped there will be free discussion by the members.

HARRY S. GRADLE.

Moved by DR. S. H. LARGE that this report be accepted and placed on file. Motion seconded and carried.

SECTION ON OPHTHALMIC AND OTO-LARYNGIC PATHOLOGY.

Again we have to report generalities. The work goes on at a fairly uniform pace. Unfortunately, not many members are taking advantage of the opportunities offered by the Army Medical Museum—not as many as are desired. It might be wise at this time to recapitulate what the Committee on Ophthalmic and Oto-Laryngic Pathology are endeavoring to do.

As you know, the Academy has joined with the Army Medical Museum in an endeavor to establish a National Museum of Ophthalmic and Oto-Laryngic Pathology. There is not enough material in the Army and Navy to bring about such a museum, consequently the Army Medical Museum, maintained in Washington under the direction of the Surgeon General of the United States Army, has offered this to the members of the Academy, particularly, and to physicians of the United States in general, to assist them in their problems of ophthalmic and oto-laryngic pathology. If a member has

a specimen of any sort, if he will send it to the Army Medical Museum, together with a description of the case from which the specimen was obtained, that specimen will be photographed, will be sectioned, will be studied histologically by the pathologists, and a report of that study with the anatomic diagnosis will be sent to the donor of that specimen. There is absolutely no cost to the donor of the specimen for this service. If it is requested, copies of the photographs and duplicate slides of the specimen will be sent the donor. Why is this done? The Museum wishes to enrich its material. It has facilities for handling material, but the amount of material available from the Army and Navy is very limited, therefore it is anxious to receive material from men in active practice.

We have received something like 600 specimens, and it was hoped to have an exhibit at this meeting, but unfortunately there was a delay and the material cannot be presented here. The past years we have been showing various wet specimens, but the loss from breakage in transit is so heavy, that we have decided to discontinue the exhibition of wet specimens, and in the future exhibit only photographs and lantern slides of actual sections. We hope in the future to make up for the lack of display this year.

The Committee can say only that it wishes more active cooperation on the part of the members of the Academy. Undoubtedly you are all throwing away a good deal of valuable pathologic material. For example, a chalazion would be a distinct addition to the museum for the purpose of study. If you have any material you do not wish to utilize, please send it to the Museum, for only in that way can we hope to have a large collection such as is necessary for a national museum on ophthalmic and oto-laryngic pathology.

HARRY S. GRADLE.

Moved by DR. LUTHER C. PETER that this report be received and filed. Motion seconded and carried.

REPORT OF THE COMMITTEE ON THE HARD OF HEARING.

Your committee begs leave to report progress in the following summary of its activities:

1. During the past year, your committee has held three meetings to consider ways and means for carrying on its work.

2. We have met in conference with members of committees of similar name and purpose of the American Otological Society, the American Laryngological, Rhinological and Oto-

logical Society, and the Section on Laryngology, Otology and Rhinology of the American Medical Association to consider means of cooperation.

3. Several of the members of your committee have become members of the Scientific Committee of the American Federation of Organizations for the Hard of Hearing, which has already done excellent foundation work in the study of problems in which we are particularly interested.

4. Your committee finds that the problems of the hard of hearing are many and present various phases. While not possessing the dramatic appeal of the deaf for their rehabilitation and meeting their social, educational and economic requirements, the outstanding feature of this problem is that of the prevention of deafness and the conservation of hearing.

5. The real remedy is to be found, in part, in a possible revision of the curriculum in many of our medical schools, to the end that every undergraduate will receive a sufficient amount of instruction in otology to make the general practitioner as proficient in the diagnosis and treatment of the commoner diseases of the ear as he is in the care and treatment of other bodily disorders.

6. A further requisite is a campaign of publicity to acquaint the public with the fundamental facts relative to the prevention of deafness, particularly with the importance of the early recognition and care of diseases tending to result in hearing loss.

7. Your committee recommends that we encourage uniform legislation in all of our States, which will provide for yearly standardized examinations of the ears of every child of school age to determine adequately its acuity of hearing with reference to the possible need of aid for existing ear disease. Pending the enactment of such legislation, your committee recommends that the public school officials of our larger cities inaugurate examinations of the acuity of hearing of all school children, in accordance with uniform standards to be determined by the Scientific Committee of the American Federations of Organizations for the Hard of Hearing.

8. At the last meeting of the American Medical Association, in Atlantic City, on the motion of one of our members, the Section on Laryngology, Otology and Rhinology voted to enlarge its committee on the Hard of Hearing by the appointment of one actively interested member in each state. At the same meeting, on the motion of one of our Fellows, a

resolution was adopted endorsing the purpose and work of the American Federation of Organizations for the Hard of Hearing. We suggest the adoption of a similar resolution by the Academy at this meeting.

9. Your committee begs leave, if it is continued, to be given authority, with the approval of the president, to change or augment its personnel at any time it may find it expedient to do so, for the sake of enlarging its usefulness as a working unit of this Academy.

10. Your committee would beg each Fellow to familiarize himself with the activities of our various agencies for the solution of the problems of the hard of hearing, and so far as he is able, in his home community, to cooperate with their activities in the carrying out of this work.

Respectfully submitted,

HOWARD V. DUTROW
HAROLD HAYS
CHAS. W. RICHARDSON
GEO. L. TOBEY
CLAUDE T. UREN
HORACE NEWHART, CHAIRMAN.

The following resolution was offered by DR. THOMAS E. CARMODY:

"RESOLVED, that the American Academy of Ophthalmology and Oto-Laryngology endorse the purposes and work of the American Federation of Organizations for the Hard of Hearing."

On motion of DR. CARMODY, duly seconded, this resolution was adopted.

REPORT OF COMMITTEE ON NECROLOGY.

During the year we have lost the following members by death:

Dr. Samuel Ellsworth Allen.....Cincinnati, Ohio
Dr. Hugo W. Aufmwasser.....Covington, Kentucky
Dr. Frank E. Auten.....Belleville, Illinois
Dr. F. E. Cheney.....Boston, Massachusetts
Dr. Claude A. Freligh.....Springfield, Mass.
Dr. William Allen Hollis.....Hartford City, Indiana
Dr. Frank Jacobi.....Toledo, Ohio
Dr. Thomas F. Keller.....Toledo, Ohio
Dr. Isaac A. Lederman.....Louisville, Kentucky
Dr. Louis F. Love.....Philadelphia, Pa.

Dr. Alexander Covington Magruder Colorado Springs, Col.
Dr. James McElroy.....Meridian, Miss.
Dr. George H. Savage.....Memphis, Tennessee
Dr. William Koenig Spiece.....Chicago, Illinois
Dr. Henry Burt Stevens.....Boston, Mass.
Dr. Robert Stevenson.....Cincinnati, Ohio
Dr. Roscoe Conkling Wilson.....Ithaca, New York

I move that we rise and bow our heads in memory of the departed, so that they, looking from above, may see that we mourn them. ARTHUR J. BEDELL.

Motion seconded and carried.

REPORT OF THE SENIOR MEMBER OF COUNCIL.

There are one or two By-Laws that require change. Section 2, Article I, where it refers to the National Board of Examiners in Oto-Laryngology, must be changed, because, since the reorganization of that Board, the name has been changed. Notice is hereby given that amendment to the Constitution will be published this year to be voted on next year.

Second, in the By-Laws, Article V, regarding dues, there has been some little possibility of variance of construction of this particular article, which has to do with the distribution of the Transactions to new members. Notice is hereby given that a proposed change will be published this year to be voted on next year.

Third, there has been no concerted action in regard to reports in local papers, either here or elsewhere. In order that some definite scheme by which this matter may be taken care of may be provided, the Council has passed the following resolution:

"RESOLVED, that matters regarding the publicity of the scientific program in local papers be controlled by the Program Committee."

The Council further recommends the approval of the report of the Committee of the American Medical Association on Compensation of Eye Injuries.

The Council has received communications from the officers of the American Medical Association concerning the revision of the laws as they now exist, or the interpretation of the laws regarding our income tax. Formerly, the item of expense in attending medical meetings was an item of deduction, but recently the interpretation of the law has been that no such deduction should be made. There is now a concerted movement

for new legislation, intelligent legislation, and to that end the following resolution has been passed:

"WHEREAS, it is the sense of the officers and Fellows of the American Academy of Ophthalmology and Oto-Laryngology, that the Income Tax Law as now administered works a hardship on physicians, in that their expenses incident to attendance on medical meetings and on postgraduate courses are not now an allowable deduction from their income; and

"WHEREAS, the narcotic tax under the Harrison Law was increased during the World War from $1.00 to $3.00; and

"WHEREAS, the necessity for this extra revenue has passed; therefore

"BE IT RESOLVED, that the Secretary of this Academy be instructed to communicate with the Secretary of the Treasury, the Chairman of the Committee on Ways and Means of the House of Representatives (Hon. William F. Green) and the Chairman of the Finance Committee of the Senate (Hon. Reed Smoot), protesting the injustice of the present laws covering these subjects; and to respectfully request them to use their good offices to further the recommendations made by the American Medical Association and other medical societies concerning the proposed legislation covering the above named subjects."

The Council would further recommend the following officers for the ensuing year:

President	E. C. Ellett	Memphis
President-elect	Ross Hall Skillern	Philadelphia
1st Vice-President	Austin A. Hayden	Chicago
2nd Vice-President	William C. Finnoff	Denver
3rd Vice-President	George L. Tobey	Boston
Treasurer	Secord H. Large	Cleveland
Secretary	Luther C. Peter	Philadelphia
Editor Transactions	Clarence Loeb	Chicago
Member Council 3 years	Samuel E. Roberts	Kansas City
Section on Instruction, 4 years	Harry S. Gradle	Chicago
American Board for Ophthalmic Examinations, 3 years	John M. Wheeler	New York
National Board of Examiners in Oto-Laryngology	Thomas E. Carmody	Denver
	William P. Wherry	Omaha

Place of meeting for 1926, Colorado Springs.

This report is respectfully submitted,

WALTER R. PARKER.

Moved by DR. THOMAS E. CARMODY that the report be adopted. Motion seconded and carried.

REPORT OF AUDITING COMMTTEE.

The Auditing Committee have found the books of the Treasurer to be correct and have certified his accounts.

ARTHUR J. BEDELL.
HARRY S. GRADLE.

On motion, duly seconded, this report was accepted.

DR. THOMAS E. CARMODY offered the following resolution:

"On account of the serious illness of our friend and past-President, Doctor Emil Mayer, I move that we extend our sincere sympathy and regret that he is unable to be with us."

On motion, duly seconded, this resolution was adopted and the Secretary instructed to communicate with Doctor Mayer.

DR. GEORGE F. KEIPER: Our President, in his Address yesterday morning, made reference to a problem that is confronting the oculists of this country, namely the activity of the optometrists, who are endeavoring by amendment of the laws in different States to force us to pass their Boards in order to be qualified to fit glasses. Dr. Patton called attention to this matter at the last meeting of the American Medical Association, and as a result a committee was appointed consisting of Drs. Patton, Zentmayer and Wescott, to take up this matter. This Academy should pay some attention to this matter. Having had some experience in legislative affairs in our State for the last twenty years, this Committee consulted me as to how to proceed. We must handle these fellows without gloves. We should organize ourselves in the different States to protect our interest, and the quickest way to get results is this—when any bill is introduced into any Legislature which will in any wise limit our perquisites in the practice of medicine, we should introduce a bill which will repeal their law, and before the Legislature adjourns they can debate the matter. I move that this Academy endorse and support this committee of the American Medical Association. (Motion seconded and carried.)

It was moved by DR. KEIPER that the Academy send greet-

ings to the American Association of Railway Surgeons, and to the American Congress on Radiology, both meeting in Chicago at this time. Motion seconded and carried.

DR. WALTER R. PARKER: I move that the amendments to the Constitution and By-Laws as published in the program, which were presented last year and published in the proper way, regarding the method of expulsion of members from the Academy, be adopted and incorporated into the By-Laws. (Motion seconded and carried.) The amendments follow:

"1. That Article 3 of the Constitution be designated as Section 1 of Article 3.

"2. That the following amendments be added to Article 3 as Section 2: 'The name of any Fellow may be dropped from the list of members if, in the unanimous opinion of Council, it is for the best interests of the Academy to take such action. Appeal may be made from action of the Council to the Academy as a whole at any annual meeting by any Fellow whose name is thus dropped. A two-thirds vote of the members present and voting will be necessary to drop a name from the list of members.'"

The following report was presented to the Oto-Laryngologic Division by the Committee on Standardization of Tuning Forks and Methods of Hearing Tests, DR. ROBERT SONNENSCHEIN, Chairman. On motion, duly seconded, this report was adopted.

REPORT OF COMMITTEE ON STANDARDIZATION OF TUNING FORKS AND METHODS OF HEARING TESTS.

To the members of the section on Oto-Laryngology of the American Academy of Ophthalmology and Oto-Laryngology:

This Committee was appointed at the meeting of the Academy in Philadelphia in October, 1921, and made reports in 1922, 1923 and 1924, with reference to the standardization of tuning forks and tuning fork tests. These are now on file in the records and Transactions of the Academy. Since there are only a few points to be added this year, we will content ourselves with a summary of the work previously presented, together with the new suggestions to be offered this year.

The committee has the following recommendations to make:

(1) Character of the fork to be employed. This should be well balanced, of rustless metal, if such can be obtained, of

pitch within a reasonable range, of fairly long duration of vibrations, moderate in price, and made so as to eliminate loud overtones, preferably by means of weights on the lower pitched forks. To avoid rattling of the weights, one manufacturer uses round, flat, clamplike devices in place of the projecting and easily broken thrumb screws. This mechanism, however, is patented, and therefore cannot be used by other manufacturers without the payment of royalty. It might be of advantage to know the "constant" of the fork, that is to say, the exact time of its decrement, and this can fairly easily be determined by the manufacturer. We do not know just how much of an item of expense this would add to the production of the fork, but unless it is very great, it would be advisable to have this "constant" determined with reference to each fork before it leaves the factory, so that otologists may know this fact, and more uniformity in forks and tests be thus effected. Very recently, we have learned through the courtesy of Dr. Harvey Fletcher (of the Bell Telephone Laboratories) that the research committee of the American Federation of Organizations for the Hard of Hearing conferred with the Bureau of Standards at Washington, D. C., who agreed to determine the "constant" of forks submitted to them. We believe our committee should get in touch with the Bureau to learn the cost of standardization, etc.

(2) The fork should be held uniformly near the ear, preferably with the wide surface of the prongs near the ear, without touching the auricle or the vibrissae, in order to get the maximum intensity of sound waves, and to avoid interference waves which occur at certain points, especially at the angle between the broad and narrow surfaces. Present the distal end of the prongs to the ear, as this gives the loudest sound per unit of area, because of the great amplitude at that point. One of the members of your Committee (Sonnenschein) has confirmed by tests with the phonometer and otherwise, that the greatest intensity of sound is obtained from the broad or outer surface of the prongs.

(3) The Committee is not yet unanimous regarding the method of stimulating the forks and the methods of timing them. We have not definitely decided whether it is better to note duration of the time during which the fork is heard, or to describe the degree of hearing by the distance from the ear at which the fork can be appreciated, using either a con-

trol or knowing the duration of the fork's vibration. However, it seems that neither method is wholly accurate; both of them should be tried out for some time before a definite conclusion is reached. To give uniform results, there should be uniform methods for the excitation of the fork. The socalled "percussion point" lies at about the junction of the distal and middle third of the prongs of the fork. It is advisable to strike the fork at this point, because one gets there the greatest intensity of sound and the least overtones. With the lowest pitched forks, such as those of from 16 to 64 double vibrations, excitation may best be had by allowing the fork to fall of its own weight, with the center of percussion or the weight (if there be one) striking the thigh, the inner edge of the hand, or the knee, care being taken to withdraw them from the object which they strike as quickly as possible, in order to prevent damping from a second contact. It is possible that using a weight in the form of a pendulum for striking the fork may give more uniform excitation, since the weight would always fall through an arc of 90 degrees, and the force of gravity would therefore give the same impact in each case. One investigator (Dr. Macfarlan) has stated that it is difficult if not impossible to get uniform results by striking the fork against the thigh, and one of the Committee (Mackenzie) is therefore opposed to this type of excitation. Instead of this method, a rubber pleximeter may be used for actuating the fork, by allowing it to fall from a perpendicular to a horizontal position, striking the "percussion point" on the broad or outer surface of one of the prongs. For the high forks such as c-4 and c-5 (respectively 2048 and 4096 double vibrations), different degrees of excitation may be used, depending upon the acuity of hearing of the patient. The lightest excitation is rubbing the prongs between the two fingers. If the sound is not heard then, strike one of the prongs with the finger nail, and if this is not heard, strike the broad surface of one of the prongs with a metal hammer. In presenting these methods of actuating the various forks, it may be noted that, generally speaking, the lower the pitch of the fork, the softer should be the object which strikes it.

When bone conduction is being tested, many examiners use their own heads, or those of other living controls. In either case, it is necessary that the otologist should know definitely whether his own or his living control's conduction

is normal, diminished, or lengthened as compared with that of the patient. One of us (Sonnenschein) has for a long time felt that a much more objective method is to determine the average duration of the time that the particular fork employed is heard by normal individuals at certain definite ages, and then compare this period with that shown by the patient in question.

(4) The Committee recommends that at first only three standard tests be considered, namely, the Weber, the Schwabach and the Rinne. For the Weber test alone, a fairly low or medium pitched fork from 100 to 256 double vibrations serves well. One of us (Mackenzie) prefers a fork of about 190 double vibrations for all of the three principal tests, "since the findings of one test can be made to serve as a check on the other two." The Weber and Schwabach tests are performed by placing the fork on the median line of the vertex or forehead, and a fork fairly heavy, so as to rest of its own weight, is desirable.

With the Weber test, see whether the sound is heard in the vertex, or, as the patient often says, "in the head;" or whether the tone of the fork is "lateralized" i.e., perceived louder or entirely in one ear or the other.

The Schwabach test is usually performed by placing the fork on the median line of the vertex or forehead, avoiding pressure except that due to the weight of the fork. Note the time that the sound is heard; occasionally lifting fork from the head to avoid the "fatigue symptom." Then as previously mentioned, compare with that of a living control using same degree of pressure, or compare it with the average duration of hearing for the particular fork used.

For the Rinne test (including the Schwabach when the bone conduction factor of the Rinne is used for this purpose) a medium pitched fork of about 400 to 500 double vibrations should be used. Mackenzie prefers one of about 190 vibrations. The time during which the fork is heard by bone and air conduction should be definitely designated. The construction of the fork should be such that the difference between the bone and air conduction is about 30 to 40 seconds. When vibrating in the air alone, the fork should be heard approximately 90 seconds. This of course requires a rather expensive fork.

A modification of the usual method of performing the Rinne test has been suggested. Ordinarily the stem of the fork is placed on the mastoid process, near the antrum; and when no longer heard, the broad surface of one of the prongs is held near the ear until the hearing by air conduction has ceased. This old technic is said to give merely the difference between, rather than a definite ratio of air to bone conduction. The modification consists in placing the stem on the mastoid for a few seconds, then removing it and holding near the ear to see whether heard by air. Then replace on the mastoid and continue this alternation every few seconds, until no longer heard either by bone or air. It would be well to try out this suggestion, to see whether the ratio obtained is a better index than the difference noted when the test is performed in the usual manner.

(5) The upper limit can be further tested either by means of the Galton (or Edelmann-Galton) whistle or the monochord. The latter is the only instrument whereby high tones can be properly tested by bone conduction.

(6) Your Committee, with the exception of Dr. Mackenzie, recommends that at least five forks (namely: C-1, 32 double vibrations; c, 64; c-1, 256; c-2, 512; and c-5, 4096 double vibrations per second) should be secured by those doing functional testing. Dr. Mackenzie prefers for low tones a fork of 64 d. v., for middle tones a fork of about 190 d. v., using this fork for Weber, Schwabach and Rinne tests, and for high tones a fork of 2,048 d. v. He aims, both with high and low tones, to test quantitatively as well as qualitatively by using a stop watch. It has been suggested to the Committee, that if we could decide definitely on the forks all of us should use, and if this idea were agreeable to the members in general, it might be possible to design forks of special shape and weight, or perhaps with a special name. This design could possibly be patented, or the name copyrighted, and the patent or copyright owned by the Society, which in turn could grant permission to any reputable manufacturer to make these forks. This would not be a burden upon those buying them, and would add a certain amount of stimulus to the purchase of the forks, besides bring about the much desired uniformity of tuning forks for ordinary tests.

(7) Although a number of efforts have been made, it has as yet been impossible to find a manufacturer who can make

forks of a rustless metal that would give as good a quality of tone and duration of vibration as the steel forks at present in use. Whether oxidized steel or a special rustless metal will prove feasible remains to be seen. However, we believe that further efforts should be made in this direction, since these forks would be of a great advantage in many ways. With the nickelled forks, it often happens that the nickelling later peels, and adventitious sounds are caused by the loose pieces of metal. On the other hand, if forks are unnickelled and are not properly oiled or otherwise protected from the air, rusting usually takes place, and this in turn will change the pitch of the fork.

(8) Some otologists are in favor of using only the c forks. Others have been accustomed to employ the Bezold A, (108 double vibration) for the Weber and Schwabach tests, and the a-1 (435 double vibrations) fork for the Rinne test. These forks have certain advantages that are hard to find in some of the other forks, but, nevertheless, they are not essential. If others are employed, the same result will be obtained in using the forks mentioned, if the same care is taken in making the tests as outlined by Bezold.

(9) With reference to the recording of tuning fork and other functional tests, some members of the Committee have for some time employed the acoumetric formula adopted by the International Congress in Buda Pesth in 1909, and have found it useful and compact. Others use the system of the Vienna Clinic, the diagram being stamped on the card and the findings noted thereon. There are others who measure the distance at which the forks are heard and express the degree of hearing in fractions, the denominator of which is the distance at which that particular fork is heard on the average by the normal ear, and the numerator the distance heard by the patient who is being examined. All of these methods are fairly accurate if done carefully and systematically, but none of them is perfect.

(10) In describing the functional tests, it is suggested that the otologist mention the patient's mental condition and the environment of the test.

(11) Considerable work has been done with audiometers of various types during the past few years. While these apparatus are no doubt of great value in obtaining audiograms which give a graphic idea of the state of hearing of the in-

dividual, still we feel that they are merely adjuncts to and cannot as yet replace the voice, tuning forks, whistles and monochord in functional tests. In the first place, the audiometers are quite expensive, and owing to the various types, no definite standardization has as yet been made. We believe further work with them should be carried on, so that within a few years there may be more uniformity in the apparatus offered. With the audiometers used at present, it is not possible to properly test bone conduction, but with tuning forks, this can very easily be done. Neither the lowest tones (below 64 and down to 16 d. v.) nor the higher tones (9,000 to 22,000 d. v.) can be tested with the office type of apparatus, but only by means of the very large, very expensive models. The hearing for the human voice (unaccentuated whisper or conversation) is the most important criterion of aural efficiency, even though it cannot well be standardized. But from a practical standpoint, sufficient data may be obtained with whisper and conversation voice without any apparatus, if simple principles are observed regarding the closing of the eyes, occlusion of the opposite ear, etc. With audiometers, so far as we are aware, voice tests cannot be made except with a phonograph attachment. Meanwhile, we would suggest that the fundamental tests with forks be carefully and systematically done with the minimum armamentarium of forks, etc., which we have mentioned.

SUMMARY: The Committee recommends first: A list of five forks to be used in ordinary work, 32, 64, 256, 512, and 4,096 double vibrations. Dr. Mackenzie recommends for the low pitched fork one between 64 and 90 double vibrations. For the middle tone, one of 190 to 200 vibrations, with which to make the Weber, Schwabach and Rinne tests. In actuating these forks, lower ones are struck with some fairly soft object, such as a rubber plexímeter or pendulum. The highest may be excited in various ways, depending upon the intensity desired, either by rubbing the prongs with the fingers, striking with the finger nail or with a metal hammer.

Second: For the highest tones, either an Edelmann-Galton whistle or the monochord should be employed.

Third: An effort should be made to investigate the feasibility of the employment of a rustless metal for the manufacture of forks.

Fourth: Further study should be made of the methods of the recording of tests and the conduction if possible of experiments with reference to distance and timing of forks in functional testing.

Supplementary Report.

Dr. Newhart, the President of the Academy, requested the Committee to formulate a simple method for the examination of school children, in the endeavor to easily separate those whose hearing is moderately or markedly impaired, from those who have normal hearing. The Committee offers the following brief procedure, in the hope that it will be of some service in the direction desired.

(a) Line up the children in rows with the eyes closed and the ears opposite to the ones being tested, firmly closed with moistened finger. Care should be taken that no part of the body such as the elbow, is in contact with the walls of the room, in order to prevent the conduction of sound by bone.

(b) Use unaccentuated whisper at a distance of six meters, employing low tones like fours, eights and nines, and high pitched ones like sixes and sevens. Segregate all those who do not hear the unaccentuated whisper at 6 meters.

(c) Test the apparently abnormal pupils, to determine their acuity of hearing for the unaccentuated whisper in each ear.

(d) Refer to the school physician all those children whose hearing for the unaccentuated whisper is less than 6 to 8 meters, so that the ears, nasopharynx and nose may be properly examined. The school physician, preferably an otologist, should then inflate with a Politzer bag having a sterilized nasal tip those cases in which there appear retracted drum membranes and diminished hearing for low pitched sounds. Then reexamine to see if improvement can be noted.

(e) The attending otologist should then continue the treatment of the cases, using his judgment about recommending the removal of infected tonsils and large or infected adenoids, if present. It is, of course, well known that many cases of conduction impairment and suppurative disease are due to the presence of adenoids.

The Committee wishes an expression of opinion from the members of the Section, as to whether the Committee should

be continued for further work or should be discharged at this time.

Respectfully submitted,
L. W. DEAN
GEO. W. MACKENZIE
E. G. GILL
ROBERT SONNENSCHEIN, CHAIRMAN.

October 22, 1925.

The first order of business Thursday morning was a report from the Senior Member of Council.

DR. WALTER R. PARKER: By the authority of Council I wish to make the following recommendations and nominations:

I wish, first, to give notice of a proposed change in the By-Laws, which refers to the Section on Instruction. The machinery for the program of the Section does not entirely cover the ground, and we wish to give notice that a change will be made. The full change will be published, but will not be voted on until next year.

Another recommendation of Council, which I wish to report to the Fellows, is that the personnel of all standing committees that do not report at the stated time should be automatically dropped, and new members put on that committee.

We have had with us as guests, this year and last year, two men of world wide reputation, and Council feels that the Academy should honor itself by nominating these men to honorary membership. I therefore place in nomination for Honorary Members of the Academy, Professor F. R. Nager, Zurich, Switzerland, and Lieut.-Colonel Henry Kirkpatrick, London, England.

Moved by DR. ARTHUR J. BEDELL that this report be accepted, and that the men named be elected to Honorary Membership in the Academy. Motion seconded and carried.

The Secretary, DR. LUTHER C. PETER, read the names of the nominees for officers of the Academy for the ensuing year (see Wednesday morning business session). On motion, duly seconded, these men were declared elected.

DR. LUTHER C. PETER: The following names have been approved by the Council, and are recommended for Fellow-

ship. These men have all qualified, and have been certified by the Ophthalmic Board or the Board on Oto-Laryngology.

Almour, Ralph W.....New York, N. Y.
Atkinson, Walter Sydney.....Watertown, N. Y.
Beyer, Arthur George.....Cincinnati, Ohio
Buchanan, Norman Duncan.....Peterborough, Ont.
Burdick, Austin F.....Lansing, Mich.
Chappell, Ralph Stephen.....Indianapolis, Ind.
Chase, Sumner B.....Fort Dodge, Iowa
Chipman, Leverett DeVebes.....St. John, N.B.
Corbett, Jeremiah J.....Boston, Mass.
Costen, James Bary.....St. Louis, Mo.
Evans, John Norris.....Brooklyn, N. Y.
Fielding, Fred G.....Glens Falls, N. Y.
Fischer, Nathaniel Arthur.....Pittsburgh, Pa.
Fisher, James A.....Asbury Park, N. J.
Fitzgerald, James P.....Chicago, Ill.
Fowler, Earl B.....Chicago, Ill.
Fowler, William.....Detroit, Mich.
Frazer, Robert Howard.....Battle Creek, Mich.
Friedenwald, Jonas Stein.....Baltimore, Md.
Fuller, Harry B.....Chicago, Ill.
Furstenberg, Albert Carl.....Ann Arbor, Mich.
Gatewood, Emmette Trible.....Richmond, Va.
Gillette, David Filsinger.....Syracuse, N. Y.
Golding, Joseph Edward.....Brooklyn, N. Y.
Hagemann, John Albert.....Pittsburgh, Pa.
Hasty, Frederick Emerson.....Nashville, Tenn.
Hofmann, William Peter.....Davenport, Iowa
Hughes, Lee Westlake.....Newark, N. J.
Jones, Charles C.....Cincinnati, Ohio
Jones, Leonard Waterhouse.....Rochester, N. Y.
Knapp, Frank Norris.....Duluth, Minn.
Knight, Mary Sedalia.....Rochester, Minn.
Kruskal, Isaac David.....Brooklyn, N. Y.
Lederer, Francis Loeffler.....Chicago, Ill.
Lifschutz, Jacob.....Chicago, Ill.
Lloyd, Ralph Irving.....Brooklyn, N. Y.
Lowry, Blackburn W.....Tampa, Fla.
Lux, Paul.....Kansas City, Mo.
McCall, Julius Watkins.....Cleveland, Ohio
McNichols, William A.....Dixon, Ill.

MacMillan, John Alexander..... Montreal, Can.
Marlow, Searle Bisset.....Syracuse, N. Y.
Messier, A. Eugene.....Worcester, Mass.
Moore, John David.....Bloomfield, N. J.
Newton, Frank Hawley.....Dallas, Texas
Noonan, William James.....Chicago, Ill.
Payne, Richard Johnson.....St. Louis, Mo.
Richardson, Shaler Arnold.....Jacksonville, Fla.
Rutherford, Cyrus Wilson.....Indianapolis, Ind.
Ryerson, Frank Leith.....Detroit, Mich.
Schlivek, Kaufman.....New York, N. Y.
Shirey, Oscar Merle.....Cleveland, Ohio
Skillern, Samuel Ruff, Jr.....Philadelphia, Pa.
Snell, A. C.....Rochester, N. Y.
Stark, Jesse B.....New York, N. Y.
Strauss, Jerome Frank.....Chicago, Ill.
Whelan, George L.....Philadelphia, Pa.

Moved by DR. PETER that the above named men be elected to Fellowship in the Academy. Motion seconded and carried.

DR. WALTER B. LANCASTER: The President, in his address, called our attention to the necessity of active publicity and cooperation. Without rehearsing what he stated, I would like to propose the organization of a standing committee, a Committee on Publicity, Service and Legislation, to cooperate with the National Committee on Publicity and Service. The National Committee consists of members from three large societies; we have a member on that committee. This National Committee does not originate actions, but matters referred to it are taken up and dealt with. We should have a committee which will not passively wait for something to turn up. Then when something which needs attention comes up, with the additional momentum of this committee, it will be referred to the National Committee. Being a standing committee, it will be appointed by the President. I move that the President appoint such a committee of two, one member to be a member of the National Committee. (Motion seconded and carried.)

THE PRESIDENT: It now gives me great pleasure to present to you one who needs no introduction to any member of the Academy, your incoming President, Dr. E. C. Ellett.

DR. E. C. ELLETT: I did not come to the platform for this purpose, but for something else which I will speak of in a moment.

For more years than I am willing to admit, I have been a regular attendant on the sessions of this Academy, and year after year I have seen the fortunate recipients of your honors get up on the platform and try to express their appreciation of election to office, but I am sure I never realized what was in their hearts or what they were trying to say. I thank you very much for this honor, and I will certainly do the best I can to carry on the affairs of the Academy as they have been in the past, and I hope when my term of office is finished, you will have no regrets at your selection.

I wish there was some way in which the Fellowship of the Academy in general could know more about the workings of your Council. We have a large Council and the attendance at this meeting has been 100 per cent.—every member of the Council being present at every meeting, and we have had several. I think you can rest easy as far as the character and the purposes of the men to whom you have entrusted the affairs of the Academy are concerned. They are faithful and earnest, and they have your interests at heart.

I came to the platform to make a motion, that the Academy express its thanks to the members of the local committee for their splendid arrangements by which they have taken care of this meeting, and for their untiring efforts in our behalf; also to the management of this hotel for the care they have taken of us under what we have all seen were most adverse circumstances.

Motion seconded and carried.

The Business Session adjourned.

ALPHABETIC LIST

Honorary Fellows

DUNDAS-GRANT, SIR JAMES...148 Harley St., W. 1, London, Eng.
ELLIOT, LT.-COL. R. H.,
54 Welbeck St., Cavendish Sq., London, W., Eng.
GRAY ALBERT A.....Glasgow, Scotland
ELSCHNIG, PROF. A., Ferdinandstrasse, 10, Prague, Czecho-Sovakia
JOHNSON, LINDSAY.....Britannia Bldg., West St., Durban, Natal
KIRKPATRICK, LT.-COL. HENRY,
54 Welbeck St., W. 1., London, Eng.
LAWFORD, J. B., 27 Weymouth St., Portland, Pl., W., London, Eng.
LISTER, SIR WILLIAM T., 24 Devonshire Place, W. 1, London, Eng.
NAGER, PROF. F.....Freiestrasse 20, Zurich, Switzerland
VAN DER HOEVE, PROF. J...Rijnsburgerweg, 6 A, Leiden, Holland

Life Members

BROWN, EDWARD J.(1911)
900 Nicollet Ave., Minneapolis, Minn., OALR
*DAYTON, W. L.....Funke Bldg., Lincoln, Neb., OALR
*FOSTER, HAL(1896)
1014 Chambers Bldg., Kansas City, Mo., ALR
HOWE, LUCIEN.....520 Deleware Ave., Buffalo, N. Y., Op.
MAYER, EMIL.....40 E. 41st St., New York City, N. Y., ALR
STANDISH, MYLES,
51 Hereford St., Back Bay, Boston, Mass., Op.
STEWART, T. M....901 Union Trust Bldg., Cincinnati, O., OALR

Fellows

ABBOTT, W. J.(1919)....8314 Euclid Ave., Cleveland, O., OALR
*ADAMS, A. L.(1905)..323 West State St., Jacksonville, Ill., OALR
ADAMS, C.....52 West State St., Trenton, N. J., OALR
ADAMS, DANIEL S.,
520 Hume-Mansur Bldg., Indianapolis, Ind., ALR
ADAMS, ELDRIDGE S.(1920)
Moore Bldg., San Antonio, Tex., OALR
ADAMS, FRANK M.(1924) 224 Thayer St., Providence, R. I., ALR
ADAMS, FRED'K W.(1920) Roosevelt Clinic, Seattle, Wash., OALR
*AGNEW, FRED'K W.(1920)
120 3rd Ave., S. E., Independence, Ia., OALR

*Attended 1925 meeting. Numbers in parenthesis indicate year in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Secretary.

AINSLEE, GEORGE (1921)
606 Oregonian Bldg., Portland, Ore., OALR
ALBRIGHT, LT. P. M., M. C. U. S. Navy, (1922)
U. S. Naval Hosp., Brooklyn, N. Y., ALR
*ALDEN, ARTHUR M.,
3858 Westminster Place, St. Louis, Mo., ALR
ALDERDYCE, W. W. (1908) ..... Nasby Blk., Toledo, O., OALR
*ALEXANDER, GEORGE JAMES (1923)
1831 Chestnut St., Philadelphia, Pa., ALR
ALKIRE, H. L. ..... 614 Kansas Ave., Topeka, Kan., OALR
*ALLEN, CHAS. E. (1920)
712 Waldheim Bldg., Kansas City, Mo., OALR
*ALLEN, G. H. (1916) ..... Mills Bldg., Topeka, Kan., OALR
*ALLEN, G. V. (1921) ..... 835 Kansas Ave., Topeka, Kan., OALR
ALLEN, J. H. ..... Majestic Bldg., Denver, Colo., ALR
ALLEN, L. P. ..... 19 Jefferson Ave., Oshkosh, Wis., OALR
ALLPORT, F. ..... 7 West Madison St., Chicago, Ill., OALR
ALMOUR, RALPH (1925) .. 51 W. 73rd St., New York, N. Y., ALR
ALTER, F. W. ..... Toledo Medical Bldg., Toledo, O., OALR
ALTRINGER, ARTHUR N.,
1100 Rialto Bldg., Kansas City, Mo., ALR
ALWAY, ROBERT D. ... 423 S. Lincoln St., Aberdeen, S. D., OALR
AMBERG, EMIL ..... David Whitney Bldg., Detroit, Mich., ALR
ANDERSON, W. B. ..... 507 Main Ave., Brownwood, Tex., OALR
*ANDREW, A. H. (1898) 30 N. Michigan Ave., Chicago, Ill., OALR
*ANDREWS, B. F. (1912) 25 E. Washington St., Chicago, Ill., OALR
ANDREWS, H. D. ..... 23 Allen St., Buffalo, N. Y., Op.
*ANDREWS, M. P. (1915) Dempsey Bldg., Manitowoc, Wis., OALR
ANNON, WALTER T. ..... 259 S. 17th St., Philadelphia, Pa., ALR
APPLEMAN, L. F. (1913)
308 South 16th St., Philadelphia, Pa., Op.
*ARBUCKLE, M. F. .... University Club Bldg., St. Louis, Mo., ALR
ARNOLD, F. J. ..... 182 Pearl St., Burlington, Vt., OALR
*ARRELL, WILLIAM ..... 317 Main, E., Hamilton, Ontario, OALR
ARROWSMITH, H. ..... 170 Clinton St., Brooklyn, N. Y., ALR
ASH, GARRETT G. ..... 1 Main St., Bradford, Pa., OALR
ASHLEY, T. W. ..... 466 Park Ave., Kenosha, Wis., OALR
ATKINSON, D. T. .. 1515 Russell Bldg., San Antonio, Tex., OALR
ATKINSON, WALTER SYDNEY (1925)
168 Sterling St., Watertown, N. Y., Op.
AUERBACH, J. ..... 120 W 86th St., New York City, N. Y., ALR
AVERY, F. T. ..... 357 W. 63rd St., Chicago, Ill., Op.LR
AYNESWORTH, H. T. (1912) ... Amicable Bldg., Waco, Tex., OALR

B

BABBITT, JAMES A. (1924)
2117 Chestnut St., Philadelphia, Pa., ALR
*BAHN, CHAS. A. (1921) Audubon Bldg., New Orleans, La., Op.
*BAILEY, F. W. (1912) ... Security Bldg., Cedar Rapids, Ia., OALR
*BAILEY, H. ..... 840 Landers Bldg., Springfield, Mo., OALR
BAIRD, CHAS. G. (1920) ..... Beatrice, Neb., OALR

BAKER, C. H.....Crapo Blk., Bay City, Mich., OALR
BAKER, H. B.(1916).....59 Main St., Taunton, Mass., OALR
BALDWIN, KATE W.....1117 Spruce St., Philadelphia, Pa., ALR
BALENTINE, P. L.(1915)
1524 Chestnut St., Philadelphia, Pa., Op.
BALL, J. MOORES.....4500 Olive St., St. Louis, Mo., Op.
BALL, M. V.....214 Penn Ave., W., Warren, Pa., OALR
*BALLENGER, H. C.....25 E. Washington St., Chicago, Ill., ALR
BALLIN, M. J.....108 E. 60th St., New York City, N. Y., ALR
BALLON, DAVID H.(1924)
255 Bishop St., Montreal, Canada, ALR
*BANE, WM.....330 Metropolitan Bldg., Denver, Colo., OALR
BANE, W. C.....330 Metropolitan Bldg., Denver, Colo., OALR
*BANISTER, J. M., Bankers Reserve Life Bldg., Omaha, Neb., OALR
BARBER, F.(1913)....75 S. Fitzhugh St., Rochester, N. Y., Op.
BARCK, C.....Humboldt Bldg., St. Louis, Mo., Op.A
BARDENHEIER, F. G. A.....Frisco Bldg., St. Louis, Mo., ALR
BARKAN, A.....Credit Swisse, Zürich, Switzerland, OALR
BARKAN, H....Medico-Dental Bldg., San Francisco, Cal., Op.
BARKER, CHAS. B.,
224½ W. Oklahoma Ave., Guthrie, Okla., OALR
BARKER, O. G. A.(1909)
Johnstown Trust Co., Johnstown, Pa., Op.
*BARLOW, ROY A.,
461 Farmers Trust Bldg., South Bend, Ind., ALR
BARNES, L. E.(1914) 6860 South Halsted St., Chicago, Ill., OALR
BARNHILL, J. F.....Pennway Bldg., Indianapolis, Ind., ALR
BARON, F. S.....Peoples Bank Bldg., Zanesville, O., OALR
BARTHOLOMEW, A. C.(1904)
122 E. Washington St., Ft. Wayne, Ind., OALR
*BASS, A. L.(1924).....Francis Bldg., Louisville, Ky., OALR
*BAUM, H. L.....Metropolitan Bldg., Denver, Colo., ALR
*BEACH, S. J.(1914).....704 Congress St., Portland, Me., Op.
BEANE, GEORGE W. .Jenkins Arcade Bldg., Pittsburgh, Pa., OALR
BEARD, H. J.(1924)...Dollar Bank Bldg., Youngstown, O., OALR
*BEATTIE, ROBERT....David Whitney Bldg., Detroit, Mich., OALR
BEATTIE, W. HENRY.....252 Genesee St., Utica, N. Y., OALR
BEATTY, H. G.....150 E. Broad St., Columbus, O., OALR
BEAUDOUX, H. A., 401 La Salle Bldg., Minneapolis, Minn., Op.A
*BECK, F. L.(1920).....408 Hynds Bldg., Cheyenne, Wyo., OALR
*BECK, J. C.(1901).....2551 N. Clark St., Chicago, Ill., ALR
BECKER, H. M.....49 S. 4th St., Sunbury, Pa., OALR
*BEDELL, A. J.....344 State St., Albany, N. Y., Op.A
*BEEBE, C. S.....Wells Bldg., Milwaukee, Wis., OALR
*BEEM, I. F.....31 N. State St., Chicago, Ill., OALR
BEGGS, W. F.....2 Lombardy St., Albany, N. Y., OALR
*BEIL, J. WALLACE(1918)
316 Argyle Bldg., Kansas City, Mo., OALR
BELL, GEO. HUSTON(1920)
40 E. 41st St., New York City, N. Y., Op.

BELL, M. H.(1913)
First Nat'l. Bank Bldg., Vicksburg, Miss., OALR
BELLIN, JOSEPH.....1026 W. 6th St., Los Angeles, Cal., OALR
BELLOWS, GEORGE E.....Rialto Bldg., Kansas City, Mo., Op.
*BENEDICT, W. L.(1919)....Mayo Clinic, Rochester, Minn., Op.
BENNETT, A. G.....26 Allen St., Buffalo, N. Y., Op.
BENNETTO, FREDERICK R.,
172 Woolwich St., Guelph, Ont., Canada, OALR
*BENTLEY, NEIL, 1161 David Whitney Bldg., Detroit, Mich., OALR
*BERENS, CONRAD....30 E. 40th St., New York City, N. Y., Op.
*BERGERON, J. Z.....104 S. Michigan Blvd., Chicago, Ill., ALR
BERNATZ, C. F.....Park Bldg., Pittsburgh, Pa., Op.
BERNSTEIN, E. J.....Professional Bldg., Detroit, Mich., OALR
BERRISFORD, PAUL D....812 Lowry Bldg., St. Paul, Minn., OALR
*BERRY, D. F.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
BETTISON, D. L.....502 Wilson Bldg., Dallas, Tex., OALR
BEYER, ARTHUR G.(1925)...19 Garfield Pl., Cincinnati, O., OALR
BEYER, LOUIS J.(1918) 449 Delaware Ave., Buffalo, N. Y., ALR
BINGER, H. E.....1027 Lowry Bldg., St. Paul, Minn., OALR
*BINKLEY, R. S.(1915)
550 Fidelity Medical Bldg., Dayton, O., OALR
BIRKETT, H. S.(1907) 252 Mountain St., Montreal, Canada, ALR
BISHOP, C. W.....616 La Salle Bldg., Minneapolis, Minn., OALR
*BLAAUW, E. E.....190 Ashland Ave., Buffalo, N. Y., OALR
BLACK, M.....Metropolitan Bldg., Denver, Colo., Op.
*BLACK, N. M.....Wells Bldg., Milwaukee, Wis., Op.
*BLACK, W. D.(1900)..Metropolitan Bldg., St. Louis, Mo., OALR
*BLACKBURN, W. J.....661 Reibold Bldg., Dayton, O., OALR
BLAIR, W. W.....Diamond Bank Bldg., Pittsburgh, Pa., Op.
BLAKE, W. F.....516 Sutter St., San Francisco, Cal., Op.
*BLAKESLEY, T. S.....Lathrop Bldg., Kansas City., Mo., OALR
*BLASSINGAME, CHARLES D.(1923)
Exchange Bldg., Memphis, Tenn., ALR
BLEDSOE, R. W.(1907) 1005 Madison Ave., Covington, Ky., OALR
*BLICKENSTAFF, A. J.(1922)
804 Peoria Life Bldg., Peoria, Ill., OALR
BLISS, C. B.....411 Columbus Ave., Sandusky, O., OALR
*BLUE, J. B.(1916).....Exchange Bldg., Memphis, Tenn., OALR
BLUM, H. N....Maison Blanche Bldg., New Orleans, La., Op.
BOBB, E. V.....1st Nat'l. Bank Bldg., Mitchell, S. D., OALR
BOECKMANN, EGIL.....Lowry Bldg., St. Paul, Minn., OALR
BOEHRINGER, H. W....1811 S. 22nd St., Philadelphia, Pa., Op
BOERNER, MORRIS H.(1923)
Scarborough Bldg., Austin, Tex., OALR
*BOILER, W. F.....426 S. Dodge St., Iowa City, Ia., OALR
BONE, H. D.....29 Parker St., Gardner, Mass., OALR
*BOONE, J. C.(1921)
411 Citizens Bank Bldg., South Bend, Ind., OALR
BOONE, JAMES L.(1924)
N. Y. Eye and Ear Inf., New York City, N. Y., ALR
*BOOT, G. W.(1909).....603 Main St., Evanston, Ill., OALR

BORDLEY, JAMES, JR. . .330 N. Charles St., Baltimore, Md., Op.
BOUCHER, R. B. . . . . .414 Banks Bldg., Vancouver, B. C., OALR
BOURBON, OLIVER P. (1920)
222 Westlake Professional Bldg., Los Angeles, Cal., OALR
BOUSQUET, J. P. E. (1924)
26 Sherbrooke St., W., Montreal, Can., OALR
BOUVY, HARRY. . . . . .New Foley Bldg., La Grange, Ore., OALR
*BOYCE, G. H. (1912) . .408 E. D. St., Iron Mountain, Mich., OALR
*BOYCE, WM. B. (1912)
1019 Ludington St., Escanaba, Mich., OALR
BOYD, E. T. (1896) . . . . . .823 Majestic Bldg., Denver, Colo., OALR
BOYD-SNEE, HARRY, 716 L. M. S. Bldg., South Bend, Ind., ALR
BOZER, HERRMANN E., 438 Delaware Ave., Buffalo, N. Y., ALR
BRADFIELD, J. A. L. . . .State Bank Bldg., La Crosse, Wis., OALR
*BRASWELL, C. J. (1922) . . . . .726 Mayo Bldg., Tulsa, Okla., OALR
BRAUNLIN, WM. H. (1920)
Marion Natl. Bank Bldg., Marion, Ind., OALR
*BRAWLEY, F. E. . . . . .30 N. Michigan Blvd., Chicago, Ill., OALR
BRAWLEY, R. V. . . . . .Wallace Bldg., Salisbury, N. C., OALR
*BRAY, E. R. . . . . .1440 Lincoln Ave., St. Paul, Minn., OALR
BRECKENRIDGE, HARRY E. . . . .209 Sixth St., Racine, Wis., OALR
BREHMER, HARRISON L. (1920)
521 Luna Blvd., Albuquerque, N. M., OALR
BREWER, JOHN D. . . . . .618 Main St., Dyersburg, Tenn., OALR
*BRIBACH, E. J. . . . . .212 Simpson Bldg., Atchison, Kan., OALR
*BRICKLEY, D. W. . . . . .196 W. Center St., Marion, O., OALR
BRIGGS, FRANCIS W. . . .400 Lyceum Bldg., Duluth, Minn., ALR
*BRIGGS, H. H. . . . . .73 Haywood St., Asheville, N. C., OALR
BRIGGS, W. E. . . . . .Box 166, Sacramento, Cal., OALR
BRITTON, G. S. . . . . .University Blk., Syracuse, N. Y., OALR
*BROBST, C. H. . . . . .Central Bank Bldg., Peoria, Ill., OALR
*BRODRICK, F. W. . . . . .511 Lawrence Bldg., Sterling, Ill., OALR
*BROOKS, E. B. . . . . .1010 Terminal Bldg., Lincoln, Neb., OALR
*BROOKS, E. H. (1915) . . .808 College Ave., Appleton, Wis., OALR
BROSE, L. D. . . . . .501 Upper First St., Evansville, Ind., OALR
BROWN, C. M. . . . . .510 Delaware Ave., Buffalo, N. Y., ALR
BROWN, E. T. . . . . .30 Church St., Burlington, Vt., OALR
BROWN, H. A. (1912)
719 St. Paul Bldg., San Francisco, Cal., Op.
*BROWN, J. E. . . . . .370 East Town St., Columbus, O., OALR
*BROWN, JOHN C. (1920) 632 Lowry Bldg., St. Paul, Minn., OALR
BROWN, J. M. . . . . .1136 W. 6th St., Los Angeles, Cal., ALR
*BROWN, L. E. . . . . .2nd Natl. Bank Bldg., Akron, O., OALR
BROWN, MORROW D., 806 Metropolitan Bldg., Denver, Colo., ALR
BROWN, MORTIMER G. (1921)
802 University Bldg., Syracuse, N. Y., ALR
BROWN, SAMUEL HORTON (1910)
1901 Mt. Vernon St., Philadelphia, Pa., Op.
*BROWN, W. E. . . . .8th Ave. and Amity St., Homestead, Pa., OALR
*BROWN, W. H.,
Bk. of Montreal Bldg., Edmonton, Alberta, Can., OALR

BRUDER, J. .....229 W. 79th St., New York City, N. Y., OALR
BRUÉRE, G. E. (1919) .....Journal Bldg., Portland, Ore., OALR
BRUMM, SETH A. (1922)
818 Stock Exchange Bldg., Philadelphia, Pa., ALR
*BRUNER, A.B. (1922) .....Guardian Bldg., Cleveland, O., Op.
BRUNER, W. E. .....Guardian Bldg., Cleveland, O., Op.
BRUST, H.O. .....720 S. Crouse Ave., Syracuse, N. Y., ALR
BRYAN, W. M. C. ....University Club Bldg., St. Louis, Mo., ALR
BRYANT, A. G. (1908) .....502 Beacon St., Boston, Mass., ALR
BUCHANAN, JAMES N. .....Tarbox Bldg., Freeport, Ill., OALR
BUCHANAN, NORMAN D. (1925)
479 Water St., Peterborough, Ont., Canada, OALR
*BUCK, R. H. .....Venetian Bldg., Chicago, Ill., OALR
BUCKLEY, J. H. .....1st Natl. Bk. Bldg., Ft. Smith, Ark., OALR
BUCKMAN, E. V. (1906)
70 S. Franklin St., Wilkes-Barre, Pa., OALR
BULL, E. L. .....124 E. State St., Ithaca, N. Y., OALR
*BULSON, A. E., JR. (1896)
406 W. Berry St.; Ft. Wayne, Ind., OALR
*BURCH, F. E. .....Hamm Bldg., St. Paul, Minn., Op.
BURDICK, AUSTIN F. (1925)
416 Tussing Blk., Lansing, Mich., OALR
BURKE, T. A. .....446 Rose Bldg., Cleveland, O., OALR
BURLESON, J. H. ....Central Trust Bldg., San Antonio, Tex., OALR
*BURNS, S. S. (1919) .....Humboldt Bldg., St. Louis, Mo., ALR
BURROUGHS, G. M. (1911) ...5 Broad St., Danielson, Conn., Op.
BURTON, F. A. .....404 Watts bldg., San Diego, Cal., ALR
BUTLER, MARGARET F., 1831 Chestnut St., Philadelphia, Pa., ALR
BUTLER, ROBERT H. (1920)
227 W. Columbus Ave., Bellefontaine, O., OALR
BUTT, M. M. .....2045 Chestnut St., Philadelphia, Pa., Op.
*BUVINGER, CHARLES W. (1921)
50 Washington St., East Orange, N. J., OALR
BYRNES, H. F. .....4 Chestnut St., Springfield, Mass., OALR

C

CAHILL, J. W. .....390 Main St., Worcester, Mass., OALR
CALDWELL, R. ....State Nat'l Bank Bldg., Little Rock, Ark., OALR
*CALHOUN, F. P. (1911) .....Candler Bldg., Atlanta, Ga., OALR
*CALLFAS, W. F. .....567 Brandeis Bldg., Omaha, Neb., ALR
CAMERON, WILLIAM G.,
1404 National Realty Bldg., Tacoma, Wash., OALR
*CAMP, W. E. ...910 Donaldson Bldg., Minneapolis, Minn., OALR
CAMPBELL, C. H.,
Columbus State Bk. Bldg., Columbus, Neb., OALR
CAMPBELL, D. A. .....David Whitney Bldg., Detroit, Mich., OALR
*CAMPBELL, D. M. .....Peter Smith Bldg., Detroit, Mich., OALR
CAMPBELL, ROBERT A.,
520 LaSalle Bldg., Minneapolis, Minn., OALR
CAMPBELL, WM. KING. .96 Third Ave., Long Branch, N. J., OALR

CAPRON, E. P.....118 Angell St., Providence, R. I., OALR
CARLETON, RALPH (1914) 10 Temple St., Springfield, Mass., Op.
*CARMACK, J. W. (1920)
505 Hume-Mansur Bldg., Indianapolis, Ind., AR
*CARMODY, T. E.....Metropolitan Bldg., Denver, Colo., ALR
*CARNCROSS, HELEN....22 E. Washington St., Chicago, Ill., Op.
CARPENTER, E. R....708 Medical Arts Bldg., Dallas, Tex., OALR
*CARPENTER, E. W.....201 E. North St., Greenville, S. C., ALR
CARR, G. W. (1909)..54 S. Franklin St., Wilkes-Barre, Pa., OALR
CARROLL, F. (1908).....Ely Blk., Cedar Rapids, Ia., OALR
*CARROLL, G. G.....614 Main St., West, Rochester, N. Y., OALR
CARSON, W. E.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
CARTER, W. W. (1896)
24 E. 48th St., New York City, N. Y., ALR
CARVILL, L. MAUD (1914)
82 Commonwealth Ave., Boston, Mass., Op.
CARY, E. H.....Southwestern Life Bldg., Dallas, Tex., OALR
CARY, G. C.....Grand Junction, Colo., OALR
*CAVANAUGH, J. A.....7 W. Madison St., Chicago, Ill., ALR
*CAYCE, E. B.....Hitchcock Bldg., Nashville, Tenn., OALR
*CHADWICK, IRA B. (1920)
A Terminal Bldg., Coffeyville, Kan., OALR
CHAMBERS, T. R. (1911)
15 Exchange Pl., Jersey City, N. J., OALR
*CHAPMAN, V. A.....Colby-Abbot Bldg., Milwaukee, Wis., OALR
CHAPPELL, RALPH S. (1925)
305 Terminal Bldg., Indianapolis, Ind., ALR
CHARLES, J. W.....Humboldt Bldg., St. Louis, Mo., Op.
*CHARLTON, C. C..1924 S. Illinois St., Atlantic City, N. J., ALR
CHASE, E. FRANK (1918) 1008 Cobb Bldg., Seattle, Wash., OALR
CHASE, SUMNER B. (1925) 630 Snell Bldg., Ft. Dodge, Ia., OALR
CHASE, WALTER D. (1924)
230 E. Broad St., Bethlehem, Pa., ALR
CHATTIN, J. FRANKLIN...671 Broad St., Newark, N. J., Op.
CHENERY, W. E...377 Commonwealth Ave., Boston, Mass., ALR
CHENEY, JAMES W....702 Schweiter Bldg., Wichita, Kan., OALR
*CHILDS, H. A.....Eagle Blk., Creston, Ia., OALR
CHIPMAN, LEVERETT DEV. (1925)
42 Coburg St., St. John, N. B., Canada, OALR
CLARK, F. T.....92 Broad St., Westfield, Mass., OALR
*CLARK, H. S.....Syndicate Blk., Minneapolis, Minn., OALR
CLARK, I. G.....188 E. State St., Columbus, O., OALR
*CLARK, J. S. (1914).....67 Stephenson St., Freeport, Ill., OALR
CLARK, L. H. (1915)...37 Monroe Ave., Rochester, N. Y., OALR
CLAY, G. E. (1917).....426 Candler Bldg., Atlanta, Ga., Op.
CLERF, LOUIS H.....128 S. 10th St., Philadelphia, Pa., ALR
CLEVENGER, W. F. (1905)
Newton Claypool Bldg., Indianapolis, Ind., ALR
COATES, G. M.....1736 Pine St., Philadelphia, Pa., ALR
COBB, EDWIN.....Masonic Temple, Marshalltown, Ia., OALR
COCHRAN, R. W. (1911) West and 3rd Sts., Madison, Ind., OALR

COFFELT, THEODORE A..Woodruff Bldg., Springfield, Mo., OALR
COGAN, J. E.....Rose Bldg., Cleveland, O., OALR
COHEN, MARTIN, 1 West 85th St., New York City, N. Y., OALR
COHEN, SAMUEL.....1626 Spruce St., Philadelphia, Pa., ALR
COHN, F.....31 East 60th St., New York City, N. Y., ALR
COHN, S.....416 Luke St., Fulton, Ky., OALR
COLLINS, F. W.....Majestic Bldg., Denver, Colo., ALR
COLLINS, H.....New Jersey Bldg., Duluth, Minn., OALR
COLLINS, J. D.....90 Main St., Northampton, Mass., ALR
*COLVER, B. N.(1913)
182 Manchester St., Battle Creek, Mich., OALR
*COMEE, W. C.....Bellin Bldg., Green Bay, Wis., OALR
CONLON, F. A.....Bay State Bldg., Lawrence, Mass., OALR
CONNOR, CHAS. E.....Hamm Bldg., St. Paul, Minn., OALR
*CONNOR, RAY(1905).....1410 Stroh Bldg., Detroit, Mich., OALR
COOK, S. E.....Richards Blk., Lincoln, Neb., OALR
*COOK, W. ALBERT(1924)...506 Palace Bldg., Tulsa, Okla., OALR
COOPER, C. E.....Metropolitan Bldg., Denver, Colo., ALR
COOVER, D. H.....Metropolitan Bldg., Denver, Colo., Op.
COPPS, LYMAN A.....Marshfield, Wis., OALR
CORBETT, JEREMIAH J.(1925)
520 Beacon St., Boston, Mass., OALR
*CORSER, J. B.....Scranton Private Hospital, Scranton, Pa., OALR
COSTEN, J. B.(1925).....316 Lister Bldg., St. Louis, Mo., ALR
*COTT, C. C.....483 Delaware Ave., Buffalo, N. Y., ALR
COTT, DELZON N.(1924).....1001 Main St., Buffalo, N. Y., ALR
COULTER, C. F.....1st Natl. Bk. Bldg., Great Falls, Mont., OALR
COVINGTON, L. C., 217½ Capitol Ave., Charleston, W. Va., OALR
COWPER, H. W.....543 Franklin St., Buffalo, N. Y., Op.
CRAFT, KENNETH L.(1921)
226 Hume-Mansur Bldg., Indianapolis, Ind., OALR
CRAIGHEAD, NANCY B.,
7002 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
CRAMPTON, G. S.....1700 Walnut St., Philadelphia, Pa., Op.
CRANE, J. WELLINGTON(1921)
128 Third St., Trenton, N. J., ALR
CREIGHTON, W. J.....1905 Chestnut St., Philadelphia, Pa., Op.
*CRISP, W. H.(1913).....Metropolitan Bldg., Denver, Col., Op.
*CROCKETT, R. L.....Oneida, N. Y., Op.
CROMAN, JOSEPH M. JR.(1924)
Washington Apts., Mount Clemens, Mich., OALR
CROSKEY, JOHN WELSH(1905)
Aldine Hotel, Philadelphia, Pa., Op.
CROSS, A. E.(1914).....Slater Bldg., Worcester, Mass., Op.
CROSS, G. H.(1919).....525 Welsh St., Chester, Pa., Op.
CROSSLEY, E. R.....29 E. Madison St., Chicago, Ill., OALR
CRUMP, J. F.....507 Citizens Bank Bldg., Pine Bluff, Ark., OALR
CULBERTSON, L. R.(1898) Masonic Temple, Zanesville, O., OALR
*CULLOM, MARVIN, McT.(1910)
208 Hitchcock Bldg., Nashville, Tenn., OALR

*Cunningham, Allan R.,
260 Barrington St., Halifax, N. S., OALR
Curdy, R. J.....1134 Rialto Bldg., Kansas City, Mo., Op.
Curran, Edw. J.(1919)
401 Waldheim Bldg., Kansas City, Mo., Op.
Curry, G. E.....Westinghouse Bldg., Pittsburgh, Pa., Op.
Curtin, Eugene A.(1923)
703 Connell Bldg., Scranton, Pa., OALR
Curtis, W. L.,
612 Security Mutual Bk. Bldg., Lincoln, Neb., OALR
*Cushman, Beulah(1924)
25 E. Washington St., Chicago, Ill., Op.
Cutler, Franklin E..1025 Schofield Bldg., Cleveland, O., ALR

D

Dabney, S. G.....Starks Bldg., Louisville, Ky., OALR
Dabney, V.(1913)
1633 Connecticut St., Washington, D. C., ALR
Dailey, Gilbert L.(1920) 713 N. 3rd St., Harrisburg, Pa., OALR
*Dancy, A. B.....First Natl. Bank Bldg., Jackson, Tenn., OALR
Daniels, R. L.....Elks Temple, New Bern, N. C., OALR
*Darmer, George A.(1921)
401 Terminal Bldg., Aurora, Ill., OALR
*Davidson, Herman P.,(1924)
22 E. Washington St., Chicago, Ill., Op.
Davies, T. Alex(1913)
578 Sherbourne St., Toronto, Can., OALR
Davis, A. E.....47 E. 57th St., New York City, N. Y., Op.
Davis, Frank C.....Lewistown, Mont., ALR
Davis, G. E.....42 W. 77th St., New York City, N. Y., OALR
*Davis, Harry B.(1920)
611 Lathrop Bldg., Kansas City, Mo., OALR
Davis, J. E.....Kress Bldg., McAlister, Okla., OALR
Davis, J. L.....135 S. 18th St., Philadelphia, Pa., ALR
Davis, Ralph F.(1920) 1104 Selling Bldg., Portland, Ore., OALR
Davis, Thomas W.,
O'Hanlon Bldg., Winston-Salem, N. C., OALR
Davis, W. Thornwall,
927 Farragut Sq., Washington, D. C., Op.
Day, Ewing W.....Westinghouse Bldg., Pittsburgh, Pa., ALR
*Dayton, G. O.....Lewisohn Bldg., Butte, Mont., OALR
*Dean, Alfred...Gilbert Blk. Bldg., Grand Rapids, Mich., Op.
Dean, F. W.....Ntl. Bank Bldg., Council Bluffs, Ia., OALR
Dean, L. W.....12 S. Clinton St., Iowa City, Ia., OALR
Dean, Walter.....Francis Bldg., Louisville, Ky., OALR
Deboe, M. P.....621 Eaton St., Key West, Fla., OALR
Decherd, H. B.....Wilson Bldg., Dallas, Tex., OALR
*Decker, J. C.(1918).....Francis Bldg., Sioux City, Ia., OALR
*Defnet, W. A.....62 Adams St., W., Detroit, Mich., ALR

DEICHLER, LYN WALLER,
2028 Chestnut St., Philadelphia, Pa., OALR
DENMAN, IRA O.....425 Ohio Bldg., Toledo, O., OALR
DENNIS, D. N.....221 W. Ninth St., Erie, Pa., Op.A
DENNIS, E. P.(1921).....311 W. 10th St., Erie, Pa., OALR
*DENNIS, F. L.....Burns Bldg., Colorado Springs, Colo., ALR
*DENNIS, G. J.(1910)...25 E. Washington St., Chicago, Ill., ALR
DERBY, G. S.....23 Bay State Rd., Boston, Mass., Op.
DETLING, F.....Investment Bldg., Los Angeles, Cal., OALR
DEWEESE, CLARENCE...154 N. Upper St., Lexington, Ky., OALR
DEWEY, C. H.(1912)
Walter Reed General Hospital, Washington, D. C.
DICKINSON, B. M.
5634 Stanton Ave., E. Liberty Sta., Pittsburgh, Pa., ALR
DINTENFASS, HENRY.....1714 Pine St., Philadelphia, Pa., ALR
DIXON, G. S.....40 E. 41st St., New York City, N. Y., R
DIXON, W. E.,
706 1st Natl. Bank Bldg., Oklahoma City, Okla., OALR
DOANE, L. L.(1905).....Reiber Bldg., Butler, Pa., OALR
*DODD, O.(1903).....708 Church St., Evanston, Ill., Op.
DOLMAN, PERCIVAL(1919) Flood Bldg., San Francisco, Cal., Op.
DONNELL, NEWMAN R.(1917)
Metropolitan Bldg., St. Louis, Mo., Op.
DONOHER, W. D.....Boston Bldg., Salt Lake City, Utah, OALR
DONOVAN, J. A.(1902).....Phoenix Bldg., Butte, Mont., OALR
DORENTO, DRED R.,
Merchants Natl. Bank Bldg., Fort Smith, Ark., OALR
DORGE, RICHARD I.(1922)
76 Seymour Ave., S. E., Minneapolis, Minn., OALR
DORSEY, J. G.....201 N. Main St., Wichita, Kan., OALR
DOUGLAS, F. A.(1922)...State Bk. Bldg., La Crosse, Wis., OALR
DOW, F. E.....4 West St., Northampton, Mass., Op.
DOWLING, J. I.....116 Washington Ave., Albany, N. Y., OALR
DOWLING, J. T.(1915).....Cobb Bldg., Seattle, Wash., OALR
DUANE, A.....139 E. 37th St., New York City, N. Y., Op.
*DUANE, J. F.(1916).....Jefferson Bldg., Peoria, Ill., OALR
DUCKWORTH, GUILFORD M.....Buchel Bldg., Cuero, Tex., OALR
DUDLEY, W. H.(1902) Brockman Bldg., Los Angeles, Cal., OALR
DULANEY, O.....717 Starks Bldg., Louisville, Ky., OALR
DUNLAP, LAWRENCE G.,
Electric Light Bldg., Anaconda, Mont., OALR
DUNN, I. J.....Masonic Temple, Erie, Pa., OALR
DUNN, O. B.....4th and Railroad Sts., Ironton, O.
DUNNINGTON, JOHN H.(1924)
104 E. 40th St., New York City, N. Y., Op.
*DUNTLEY, G.S.(1913)....220 E. Carroll St., Macomb, Ill., OALR
*DUTROW, H. V...1040 Fidelity Medical Bldg., Dayton, O., OALR
DUTTON, C. K.....Moberly, Mo., OALR
DWIGHT, C. G.....Pioneer Bldg., Madison, Wis., OALR
DWORETZKY, JULIUS.....7 Law St., Liberty, N. Y., ALR
DYER, CLYDE P.(1920) Metropolitan Bldg., St. Louis, Mo., Op.

E

*Earel, A. M.....507 Wildon Blk., Hoopeston, Ill., OALR
Earl, Harry D.....3717 Spruce St., Philadelphia, Pa., ALR
Easton, J. C.(1904).....Fairbanks Bldg., Springfield, O., OALR
Edson, R.....560 Delaware Ave., Buffalo, N. Y., Op.
*Edwards, C. J.....1422 Wash St., Vicksburg, Miss., OALR
*Edwards, S. R.(1918)
111 N. Lafayette St., South Bend, Ind., OALR
Edwards, W. A.....329 Main St., La Crosse, Wis., OALR
Ellegood, J. A.....Equitable Bldg., Wilmington, Del., OALR
*Ellett, E. C.(1899)...Exchange Bldg., Memphis, Tenn., Op.
Elwood, Calvin R.(1922) Spies Bldg., Menominee, Mich., OALR
Emerson, L.(1906)....Metropolitan Bldg., Orange, N. J., OALR
*Engle, H. P.....200 Olive St., Newton, Ia., OALR
Ersner, Matthew S....1729 Pine St., Philadelphia, Pa., ALR
Estabrook, C. T.....390 Main St., Worcester, Mass., OALR
Esterly, D. E.....Mills Bldg., Topeka, Kan.
Evans, John N.(1925)
23 Schermerhorn St., Brooklyn, N. Y., Op.
Evensen, H. O.....25 E. Washington St., Chicago, Ill., OALR
Ewing, Arthur E...Metropolitan Bldg., St. Louis, Mo., Op.

F

Fairbairn, John.....503 Delaware Ave., Buffalo., N. Y., ALR
*Fairchild, Nora M.(1922)
600 Brandeis Theater Bldg., Omaha, Neb., Op.
Fairing, John W.(1924)
204 Tribune Bldg., Greensburg, Pa., OALR
*Faith, Thomas.....31 N. State St., Chicago, Ill., OALR
Fallows, H. D.(1916)
Park Hospital Clinic, Mason City, Ia., OALR
*Farmer, A. G.....1040 Fidelity Med. Bldg., Dayton, O., OALR
Farnesworth, M. A.
146 Greenwood St., Battle Creek, Mich., OALR
Farnsler, H. H.....1438 Market St., Harrisburg, Pa., OALR
Farrell, P. J.....25 E. Washington St., Chicago, Ill., OALR
*Farrington, P. M...1473 Goodbar Ave., Memphis, Tenn., ALR
*Fawcett, I.....Schmulbach Bldg., Wheeling, W. Va., OALR
*Fellows, G. G.....30 N. Michigan Blvd., Chicago, Ill., OALR
Felt, Carl L.,
N. E. Cor. Chestnut & 20th Sts., Philadelphia, Pa., ALR
*Fenton, Ralph A.....616 Journal Bldg., Portland, Ore., OALR
*Ferguson, E. I.(1904)
607 1st Natl. Bank Bldg., Oklahoma City, Okla., OALR
Ferman, John W.(1921)..Stahlman Bldg., Charleroi, Pa., OALR
Fernandez, Francisco M.....Prado 105, Havana, Cuba, OALR
Fielding, Fred G.(1925) 72 Ridge St., Glens Falls, N. Y., ALR
*Finnoff, W. C.(1916).....Imperial Bldg., Denver, Colo., Op.
Fischer, Nathaniel A.(1925)
623 Union Trust Bldg., Pittsburgh, Pa., ALR

FISCHER, V. B.....Mercantile Bk. Bldg., Boulder, Colo., OALR
FISHER, CARL..1028 Pacific Mut. Bldg., Los Angeles, Cal., Op.A
FISHER, JAMES A.(1925)
Kinmouth Bldg., Asbury Park, N. J., OALR
*FISHER, WM. A.....31 N. State St., Chicago, Ill., OALR
FISKE, G. F.....25 E. Washington St., Chicago, Ill., OALR
FITZGERALD, JAMES P.(1925)
104 S. Michigan Blvd., Chicago, Ill., Op.
FLAGG, J. D.(1900).....473 Virginia St., Buffalo, N. Y., Op.A
*FLATLEY, T. J.....Reliance Bldg., Moline, Ill., OALR
*FLOYD, T. W.(1918).....Lehman Bldg., Peoria, Ill., OALR
FLYNN, JAMES A..1616 Eye St., N. W., Washington, D. C., ALR
*FOGARTY, CHAS. W....1826 Summit Ave., St. Paul, Minn.,OALR
FOLEY, J. W(1915).....Majestic Bldg., Denver, Colo., OALR
FORBES, H. H.....40 E. 41st St., New York City, N. Y., ALR
*FORBES, S. B.....409 Citizens Bk. Bldg., Tampa, Fla., OALR
*FORGRAVE, L. ROBERT.....Logan Bldg., St. Joseph, Mo., OALR
FORSYTHE, S. T.(1919)...1011 Euclid Ave., Cleveland, O., OALR
FORT, A. G.....833 Candler Bldg., Atlanta, Ga., OALR
FOSTER, J. H.....1300 Walker Ave., Houston, Tex., ALR
FOSTER, J. M.....Metropolitan Bldg., Denver, Colo., OALR
*FOUTS, JOHN D...1110 Fidelity Medical Bldg., Dayton, O., OALR
FOWLER, EARL B.(1925).....31 N. State St., Chicago, Ill., Op.
FOWLER, S. R.....University Bldg., Syracuse, N. Y., ALR
FOWLER, WILLIAM(1925)
808 Empire Bldg., Detroit, Mich., OALR
*FOX, CHAS. H.(1920)...2116 Central Ave., Kearney, Neb., OALR
FOX, L. W.....303 S. 17th St., Philadelphia, Pa., Op.
FOX, R. D.....411 Unity Bldg., Bloomington, Ill., OALR
FRANCHERE, FRED'K E.(1920)
4th and Nebraska Sts., Sioux City, Ia., OALR
*FRANCIS, LEE M.....636 Delaware Ave., Buffalo, N. Y., Op.
FRANK, A. C....308 Diamond Bank Bldg., Pittsburgh, Pa., ALR
FRANK, IRA(1908)....104 S. Michigan Blvd., Chicago, Ill., ALR
FRAZER, ROBERT H.(1925)
14 Greenwood Ave., Battle Creek, Mich., ALR
FREEDMAN, L. M.(1920)..419 Boylston St., Boston, Mass., ALR
*FRENCH, R. F...Masonic Temple Bldg., Marshalltown, Ia., OALR
FREUDENTHAL, W..24 W. 88th St., New York City, N. Y., ALR
FREY, C. L.....529 Vine St., Scranton, Pa., OALR
FRIDENBERG, P. H...38 W. 59th St., New York City, N. Y., OALR
FRIEDENWALD, H.....1029 Madison Ave., Baltimore, Md., Op.A
FRIEDENWALD, JONAS S.(1925)
1212 Eutaw Pl., Baltimore, Md., Op.
FRIEDMAN, D.....44 W. 77th St., New York City, N. Y., ALR
*FRINGER, W. R.....Wm. Brown Bldg., Rockford, Ill., Op.
FULKERSON, C. B.,
Kalamazoo Natl. Bank Bldg., Kalamazoo, Mich., OALR
*FULLENWIDER, C. M.....Barnes Bldg., Muskogee, Okla., OALR
FULLER, HARRY B.(1925)....31 N. State St., Chicago, Ill., OALR

*Fuller, T. E.,
Texarkana Natl. Bank Bldg., Texarkana, Tex., OALR
Fulton, John E.....728 Lowry Bldg., St. Paul, Minn., OALR
Furgason, A. P.....Richards Blk., Lincoln, Neb., OALR
Furstenberg, Albert C.(1925)
330 S. State St., Ann Arbor, Mich., ALR
Fust, John H.(1922).....138 W. 9th St., Erie, Pa., OALR

G

*Gadbois, Arthur E.(1921)
421 Norfolk Ave., Norfolk, Neb., OALR
*Gailey, William W.(1923)
615 Griesheim Bldg., Bloomington, Ill., OALR
Gale, M. Jean(1919).....Imperial Bldg., Denver, Colo., Op.
Gallaher, T. J.....California Bldg., Denver, Colo., ALR
Garber, J. M.....48 Park Ave., Mansfield, O., OALR
*Garraghan, E. F.....25 E. Washington St., Chicago, Ill., OALR
Garrett, J. R.(1913).....Strickland Bldg., Roanoke, Va., OALR
Gatewood, Emmette T.(1925)
Professional Bldg., Richmond, Va., ALR
Gatewood, W. L....15 E. 48th St., New York City, N. Y., ALR
Gavin, E. F.....1 S. Sheridan Rd., Waukegan, Ill., OALR
Geiger, C. W.(1904).....Cobb Bldg., Kankakee, Ill., OALR
George, H. W.....19 N. Union St., Middletown, Pa., Op.
Gibson, R. D....Dollar Savings & Trust, Youngstown, O., OALR
Gifford, H.....Brandeis Bldg., Omaha, Neb., Op.A.
*Gifford, Sanford R.(1919)
702 Brandeis Theater Bldg., Omaha, Neb., Op.
Gilbert, F. Y.....148 Park St., Portland, Me., Op.AR
Gill, E. G.(1919).....McBain Bldg., Roanoke, Va., ALR
Gill, G.....146 Middle Ave., Elyria, O., OALR
Gill, G. Bache(1923)...1624 Eye St., Washington, D. C., ALR
Gillett, Wilbur G.....201 N. Main St., Wichita, Kas., Op.
Gillette, David F.(1925)...811 Park St., Syracuse, N.Y., Op.
*Gillum, John R.(1920) 221 S. 6th St., Terre Haute, Ind., OALR
Gittens, T. R.....Sioux City, Ia., OALR
Glasford, Samuel T.(1924).....Pekin, Ill., OALR
Gleason, E. B.....2033 Chestnut St., Philadelphia, Pa.,OALR
Glogau, O.....64 E. 91st St., New York City, N. Y., OALR
Glosser, H. H.(1912)....448 Franklin St., Buffalo, N. Y., OALR
Glover, S. P.....1118 Twelfth Ave., Altoona, Pa., OALR
Goddard, H. M.....1531 Spruce St., Philadelphia, Pa., ALR
Goldbach, L. J.(1911)
1012 N. Charles St., Baltimore, Md., OALR
Golding, Joseph E.(1925)
170 Hancock St., Brooklyn, N. Y., Op.
*Goldman, Harry G....57 W. 58th St., New York, N. Y., ALR
Goldsmith, P. G.....84 Carlton St., Toronto, Can., ALR
*Goldstein, M. A....3858 Westminster Pl., St. Louis, Mo., ALR
*Goodyear, Henry M.(1924)
3029 Fairfield Ave., Cincinnati, O., ALR

*GORDAN, J. S.....309 Goldsmith Bldg., Milwaukee, Wis., OALR
*GORRELL, T. J.,
Vincennes and 16th Sts., Chicago Heights, Ill., OALR
GOSNEY, CHAS. W.(1920)
6075 Franklin Ave., Los Angeles, Cal., OALR
GOTTLIEB, M. J.814 West End Ave., New York City, N. Y., ALR
GOURLEY, G. F.(1917).407 Sinclair Bldg., Steubenville, O., OALR
*GOUX, R. S.....545 David Whitney Bldg., Detroit, Mich., OALR
*GRACEY, GEORGE F.....209 State St., Harrisburg, Pa., OALR
*GRADLE, HARRY S.(1911)
22 E. Washington St., Chicago, Ill., Op.
GRAHAM, H. B.....210 Post St., San Francisco, Cal., ALR
*GRANGER, WAYNE B.(1920)
705 Commercial St., Emporia, Kas., OALR
*GRANT, F. E.(1912)
Kal. Natl. Bank Bldg., Kalamazoo, Mich., OALR
*GRANT, P. T.,
205 Widdicomb Bldg., Grand Rapids, Mich., OALR
GRAVES, W. H.....Commerce Bldg., Pittsburg, Kan., OALR
GREEN, A. S.....853 Hyde St., San Francisco, Cal., Op.
GREEN, J., JR.....Metropolitan Bldg., St. Louis, Mo., Op.
GREEN, L. D.....853 Hyde St., San Francisco, Cal., Op.
*GREENE, JOS. B.(1923)...Haywood Bldg., Asheville, N. C., ALR
GREENWOOD, A....82 Commonwealth Ave., Boston, Mass., Op.
GREEVER, BOYD L.,
309 State Exchange Bldg., Hutchinson, Kan., OALR
GREGG, JOHN B.,
Security Natl. Bank Bldg., Sioux Falls, S. D., OALR
GRIM, U. J.....30 N. Michigan Blvd., Chicago, Ill., OALR
GRISCOMB, J. MILTON..1925 Chestnut St., Philadelphia, Pa., ALR
GROSS, JULIUS E.(1920)
8030 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
GROSVENOR, L. N.....378 Dakota St., Huron, S. D., OALR
*GROVE, W. E.(1914)...1330 Wells Bldg., Milwaukee, Wis., OALR
GRUSHLAW, I..271 Central Pk., W., New York City, N. Y., ALR
GSELL, J. F.(1920).....911 Beacon Bldg., Wichita, Kas., OALR
GUERINOT, ALBERT J.(1918)
7094 Jenkins Arcade Bldg., Pittsburgh, Pa., OALR
GUGGENHEIM, L. K.....Carleton Bldg., St. Louis, Mo., ALR
*GUNDELACH, C. A.,
825 University Club Bldg., St. Louis, Mo., ALR
GUTHRIE, A. L.(1910)
Amer. Natl. Bank Bldg., Oklahoma City, Okla., OALR
GUTHRIE, J. M.(1912).Rosenbaum Bldg., Meridian, Miss., OALR
GUTTMAN, J....987 Madison Ave., New York City, N. Y., OALR
*GUYTON, B. S.....Carter Bldg., Oxford, Miss., OALR
GWINN, G. E.(1916)
704 Russell Bldg., San Antonio, Tex., OALR

H

*HAAS, FRED'K J.....Masonic Bldg., Leavenworth, Kas., OALR

HACKNEY, F. J. .630 Volunteer Bldg., Chattanooga, Tenn., OALR
HADEN, H. C. . .913 Union Natl. Bk. Bldg., Houston, Tex., OALR
HAGEMAN, JOHN A.(1925) Highland Bldg., Pittsburgh, Pa., ALR
*HAGER, W. A. . . . .111 N. Lafayette St., South Bend, Ind., OALR
HAGLER, A. L. . . . . .Hagler Bldg., Springfield, Ill., OALR
HAGLER, E. E.(1896) . . . . . .Hagler Bldg., Springfield, Ill., OALR
*HALL, ALICE K.(1924) .25 E. Washington St., Chicago, Ill., ALR
*HALL, EDW. P. . . . . .Syracuse, N. Y., OALR
HALL, G. C.(1907) . . . . . .Gaulbert Bldg., Louisville, Ky., OALR
HALSTED, F. S. . . . .708 Metropolitan Bldg., Denver, Colo., OALR
HAMPTON, R. R. . . . . .Boston Bldg., Salt Lake City, Utah, OALR
HANCOCK, J. C.(1914)
135 Cambridge Pl., Brooklyn, N. Y., Op.
*HANEY, WM. P.,
703 Brandeis Theater Bldg., Omaha, Neb., OALR
HANS, C. L.(1921) . . . . . .19 W. 7th St., Cincinnati, O., OALR
HANSEN, ERLING W.,
910 Donaldson Bldg., Minneapolis, Minn., OALR
HARBERT, J. P.(1918) . .135 N. Ulain St., Bellefontaine, O., OALR
*HARDESTY, J. F.(1920)
311 Metropolitan Bldg., St. Louis, Mo., Op.
HARDY, W. F.(1914) . . . . .Humboldt Bldg., St. Louis, Mo., Op.
*HARKNESS, G. F. . . . . .2130 Main St., Davenport, Ia., OALR
*HARRIS, C. M.(1910) Johnstown Trust Co., Johnstown, Pa., OALR
*HARRIS, H. B. . . . . .Fidelity Med. Bldg., Dayton, O., OALR
HARRIS, W. C. . . . . .Groton Bldg., Cincinnati, O., ALR
*HARRIS, W. ROBIN. . . . . .Exchange Bldg., Memphis, Tenn., ALR
HARRISON, W. G. . . . . .Empire Bldg., Birmingham, Ala., OALR
HARTLEY, F. A. . . . . .Fairbanks Bldg., Springfield, O., OALR
HARTZ, H. J. . . . . .27 E. Adams St., Detroit, Mich., ALR
HARTZEL, SOL. M. . . . .Dollar Bank Bldg., Youngstown, O., OALR
HARVEY, N. D. . . . . .114 Waterman St., Providence, R. I., OALR
HASKELL, A. D. . . . . .145 High St., Portland, Me., Op.
HASKIN, W. H.(1913) . . .40 E. 41st St., New York, N. Y., ALR
*HASPEL, D. M.(1911)
1214 Maison Blanche Bldg., New Orleans, La., OALR
HASTY, FREDERICK E.(1925)
420 Lambuth Bldg., Nashville, Tenn., OALR
HATCH, C. B.(1908) . . . . . .3 W. Church St., Newark, O., OALR
*HAUER, A. M. . . . . .327 E. State St., Columbus, O., OALR
*HAUGHEY, W.(1911) .24 W. Main St., Battle Creek, Mich., OALR
HAWLEY, C. W. . . . . .30 N. Michigan Blvd., Chicago, Ill., OALR
HAWMAN, ERLE G. . . .4051 N. Broad St., Philadelphia, Pa., OALR
*HAYDEN, AUSTIN A. . . .25 E. Washington St., Chicago, Ill., OALR
*HAYS, H. N.(1912) .22 W. 47th St., New York City, N. Y., ALR
*HAZLETT, L. R. . . . . .117 S. Main St., Butler, Pa., OALR
HEALEY, JOHN F. . . . . .503 Delaware Ave., Buffalo, N. Y., ALR
HEARD, C. F. . . . . .Masonic Temple Bldg., Erie, Pa., OALR
HEARD, MARY K. . . . . .41 W. Main St., North East, Pa., Op.
HEATH, PARKER(1924)
David Whitney Bldg., Detroit, Mich., Op.

*HECKEL, E. B.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*HEEB, H. J.....Loan & Trust Bldg., Milwaukee, Wis., OALR
HEED, C. R.....1205 Spruce St., Philadelphia, Pa., Op.
HEGGIE, N. M. (1905)...Buckman Bldg., Jacksonville, Fla., OALR
*HEITGER, J. D. (1905)...808 Francis Bldg., Louisville, Ky., OALR
HEMPSTEAD, BERT E.....Mayo Clinic, Rochester, Minn., ALR
HENNESSEY, WM. W.....333 Essex St., Salem, Mass., OALR
HENNING, CARL.....The Rochambeau, Washington, D. C., Op.
HENNINGER, L. L...205 Citizens Bk. Bldg., Pasadena, Cal., OALR
HENTON, GEO. E. (1918).412 Morgan Bldg., Portland, Ore., OALR
HERBERT, A. W....119 E. Lehigh Ave., Philadelphia, Pa., OALR
HERRIMAN, L. L. (1918).Physicians Bldg., Alamosa, Colo., OALR
HESS, W. L.....1554 California St., Denver, Colo., OALR
*HETRICK, LLEWELLYN E.,
20 East 53d St., New York, N. Y., ALR
HETZEL, C. C.....Eccles Bldg., Ogden, Utah, OALR
HIGBEE, E. H. (1908).Grand and Olive Sts., St. Louis, Mo., Op.
HIGGINBOTHAM, T. L.,
Solomon Clinic Bldg., Wichita, Kas., OALR
HIGGINS, REUBEN P. (1924)..20 Court St., Cortland, N. Y., OALR
*HIGGINS, S. C.....Wells Bldg., Milwaukee, Wis., OALR
HILL, A. J. (1910)..301 Citizens Natl. Bk. Bldg., Salem, O., OALR
*HILL, CLARENCE E. (1919)...160 Bloor St., Toronto, Can., Op.
*HILL, EMORY.....Professional Bldg., Richmond, Va., Op.
*HINMAN, EUGENE E.,
146 Washington Ave., Albany, N. Y., ALR
HINNEN, G. A.....1343 Delta Ave., Cincinnati, O., OALR
HITZ, H. B.....Goldsmith Bldg., Milwaukee, Wis., ALR
HOBART, BERTHA KING..304 Pro. Exch. Bldg., Toledo, O., OALR
*HODSDON, B. F.....25 N. E. First Ave., Miami, Fla., OALR
*HOFFMAN, WALTER F....916 Leary Bldg., Seattle, Wash., OALR
HOFMANN, WILLIAM P. (1925).....Davenport, Ia., OALR
HOGAN, WALTER L. (1924)..750 Main St., Hartford, Conn., OALR
HOGSHEAD, J. M....602 Georgia Ave., Chattanooga, Tenn., OALR
HOGUE, D. W. (1908).....Fairbanks Bldg., Springfield, O., OALR
HOGUE, G. I.....410 Jefferson St., Milwaukee, Wis., OALR
*HOHF, J. A. (1915).....Clinic Bldg., Yankton, S. D., OALR
HOLDSWORTH, FRANK,
206 Wilhelm Blk., Traverse City, Mich., OALR
HOLLOWAY, T. B.....1819 Chestnut St., Philadelphia, Pa., Op.
HOLMES, BENJ. H.....432 Baker Bl., Racine, Wis., OALR
HOLT, E. E., JR.....823 Congress St., Portland, Me., Op.A
*HOMPES, J. J.....Security Mutual Bldg., Lincoln, Neb., OALR
*HOOD, T. C.....Hume-Mansur Bldg., Indianapolis, Ind., Op.
HOPKINS, W. E....515 Park Ave., New York City, N. Y., OALR
HOPPER, WILBUR L.....209 S. Main St., Fort Scott, Kan., OALR
HOTCHKISS, W. W....195 Forest Ave., Jamestown, N. Y., OALR
*HOURN, GEORGE E. (1920)
703 University Club Bldg., St. Louis, Mo., ALR
*HOVDE, A. G. (1916).Board of Trade Bldg., Superior, Wis., OALR
*HOWARD, C. N. (1919).....Warsaw Clinic, Warsaw, Ind., OALR

HOWARD, E. F.(1919)
First Natl. Bank Bldg., Vicksburg, Miss., ALR
HOWARD, EDWARD L.....First Natl. Bk., Ft. Worth, Tex., OALR
HOWARD, J. W.....Rialto Bldg., Kansas City, Mo., OALR
*HOWARD, LLOYD G.....Council Bluffs, Iowa, OALR
HOWARD, WILLIAM H.,
137 N. Fairview Ave., St. Paul, Minn., OALR
HOWE, ARCHIBALD W.,
Puget Sound Bk. Bldg., Tacoma, Wash., OALR
HUBBARD, A. E.....372 Franklin St., Bufftlo, N. Y., Op.
HUGHES, LEE W.(1925)...1019 Broad St., Newark, N. J., OALR
HUGHES, THOMAS E.(1922)
Medical Arts Bldg., Richmond, Va., ALR
*HUGHES, W. F....Hume-Mansur Bldg., Indianapolis, Ind., Op.
HUNTINGTON, ROBT. H.....Eureka Springs, Ark., OALR
HURD, L. M.(1910)..39 E. 50th St., New York City, N. Y., ALR
HURFF, J. W.(1919)...86 Washington St., Newark, N. J., OALR
HURLEY, E. D.(1914).....419 Boylston St., Boston, Mass., Op.
HURLEY, E. H.....419 Boylston St., Boston, Mass., Op.
HURST, V. REEVES.....Longview, Tex., OALR
HUSIK, DAVID N.(1921).1610 Spruce St., Philadelphia, Pa., ALR
*HUSSEY, E. J.....276 High St., Holyoke, Mass., Op.LR
HUTCHINSON, O. G.....Schweiter Bldg., Wichita, Kas., OALR

I

*IGLAUER, S.....Livingston Bldg., Cincinnati, O., ALR
IMPERATORI, C. J....17 E. 38th St., New York City, N. Y., ALR
INGERMAN, S. M.(1909)
418 Central Park, W., New York City, N. Y., OALR
*INGERSOLL, J. M.(1908).....Osborn Bldg., Cleveland, O., ALR
INGLIS, H. J.....43 Bay State Bldg., Boston, Mass., ALR
*INGRAM, L. C.(1915).....Clinic Bldg., Olanda, Fla., OALR
IRVIN, EDGAR H.....321 W. Rio Grande St., El Paso, Tex., OALR
IRVINE, A. R.(1918)
709 Westlake Professional Bldg., Los Angeles, Cal., Op.
IRWIN, F. N.....114 E. 54th St., New York City, N. Y., OALR
IRWIN, V. J.....389 Main St., Springfield, Mass., OALR
*IRWIN, VINCENT J., JR.(1921)
274 Main St., Springfield, Mass., OALR
*ISAACS, D.....Brandeis Theater Bldg., Omaha, Neb., OALR
ISRAEL, J. P...U. S. Veterans Bureau, San Francisco, Cal., OALR
*ISRAEL, N. E.....404 Carter Bldg., Houston, Tex., OALR
*ISRAEL, S.....Carter Bldg., Houston, Tex., OALR
*IVINS, H. M.(1910)...434 Higley Bldg., Cedar Rapids, Ia., OALR

J

JACKLE, ARTHUR F.....63 E. Fourth St., Dunkirk, N. Y., Op.
JACKSON, C.....128 S. 10th St., Philadelphia, Pa., L
*JACKSON, E.(1902).....Imperial Bldg., Denver, Colo., Op.
JACKSON, J. D.(1920).....2324 West 8th St., Erie, Pa.

*JACOBS, M. W.....Carleton Bldg., St. Louis, Mo., Op.
JACOBS, S. M....1018 E. 163rd St., New York City, N. Y., OALR
JACOBY, D. P. A.(1912)....106 Touro St., Newport, R. I., Op.AL
JAMES, J. H.....Natl. City Bank Bldg., Mankato, Minn., OALR
JARECKY, H.....138 W. 86th St., New York City, N. Y., OALR
JEAN, GEORGE W...San Marcos Bldg., Santa Barbara, Cal., Op.
JENNINGS, J. E.....Carleton Bldg., St. Louis, Mo., OALR
*JESSAMAN, L. W.(1914)
182 Concord St., Framingham, Mass., OALR
*JOBSON, G. B.(1912).....4 Printz Bldg., Franklin, Pa., OALR
*JOHNSON, A. E.(1919)
Citizens Bank Bldg., Watertown, S. D., OALR
JOHNSON, H. R.....512 Walnut Ave., Fairmont, W. Va., OALR
JOHNSON, OTIS H.,
630 Volunteer Bldg., Chattanooga, Tenn., OALR
JOHNSON, THOMAS H.,
30 W. 59th St., New York City, N. Y., Op.
*JOHNSTON, W. H.(1912).....Layrel Bldg., Muscatine, Ia., OALR
*JOHNSTON, WILSON.....Stevens Bldg., Portland, Ore., OALR
*JONES, CHARLES C.(1924)...19 Garfield Pl., Cincinnati, O., ALR
JONES, E. L.(1913)
First Natl. Bank Bldg., Cumberland, Md., OALR
JONES, E. W.....44 Atlanta St., Lynn, Mass., Op.A
*JONES, F. W.....Times Bldg., Alton, Ill., OALR
JONES, LEONARD W.(1925)
53 S. Fitzhugh St., Rochester, N. Y., OALR
*JORDAN, G. T.....30 N. Michigan Blvd., Chicago, Ill., OALR
JOYES, CRITTENTON,
Farmers & Merch. Natl. Bk. Bldg., Ft. Worth, Tex., OALR
*JOYNT, ALBERT J.(1924).....Black Bldg., Waterloo, Iowa, OALR
JUDD, HAROLD B.(1917)
47 E. 57th St., New York City, N. Y., ALR
JUDGE, HARRY V.(1920).....46 Willett St., Albany, N. Y., Op.

K

KAHN, HARRY.....104 S. Michigan Blvd., Chicago, Ill., ALR
*KAHN, MYRON E.....29 E. Madison St., Chicago, Ill., ALR
*KAUFFMAN, ARNOLD B.(1922)
305 Gurney Bldg., Syracuse, N. Y., ALR
KAUFMAN, A. S.....1923 Spruce St., Philadelphia, Pa., ALR
KEARBY, D. O..422 Am. Cen. Life Bldg., Indianapolis, Ind., ALR
KEELER, J. C.....254 S. 16th St., Philadelphia, Pa., ALR
KEIM, WILLIAM F.(1924)..2 Lombardy St., Newark, N. J., OALR
*KEIPER, G. F.(1896).....14 N. Sixth St., Lafayette, Ind., OALR
KEITH, D. M.....414 N. Main St., Rockford, Ill., ALR
KEITH, W. E.....807 Waldheim Bldg., Kansas City, Mo., OALR
KELLEY, C. W., JR.....328 Francis Bldg., Louisville, Ky., OALR
KEMLER, JOSEPH J.(1922).1908 Eutaw Pl., Baltimore, Md., OALR
KENDALL, M. R.....614 Osborn Bldg., Cleveland, O., ALR
KENNEY, WM. L.....Corby Forsee Bldg., St. Joseph, Mo., OALR

*KENNON, WILLIAM G.(1924)
Lambuth Bldg., Nashville, Tenn., OALR
KERRICK, STANLEY E..407 Security Bldg., Hollywood, Cal., OALR
KERRISON, P. D....58 W. 56th St., New York City, N. Y., ALR
KEY, B. W.(1914)...7 W. 49th St., New York City, N. Y., Op.
KEYS, MORTON J...307 Jones Blk., Victoria, B. C., Canada, OALR
KIEFER, H. A.....406 Brockman Bldg., Los Angeles, Cal., OALR
KIEHLE, FRED'K A.....1000 Corbett Bldg., Portland, Ore., OALR
*KILBOURNE, PERLEY H.(1924)
Fidelity Med. Bldg., Dayton, O., OALR
*KIMBERLIN, J. W.....900 Rialto Bldg., Kansas City, Mo., Op.
*KINCAID, J. H.....Ross Flats, Knoxville, Tenn., OALR
*KING, C.(1910).....Union Central Bldg., Cincinnati, O., Op.
*KING, CHARLES R.(1920)...471 Spitzer Bldg., Toledo, O., OALR
*KING, EDWARD.....605 Traction Bldg., Cincinnati, O., ALR
*KING, G. I.....537 E. Market St., Alliance, O., OALR
KING, J. J.....40 E. 41st St., New York City, N. Y., ALR
KING, O. H.....Dugan Stuart Bldg., Hot Springs, Ark., OALR
KIRBY, DANIEL B.(1913)
30 W. 59th St., New York City, N. Y., Op.
KIRKENDALL, J. S.....315 N. Aurora St., Ithaca, N. Y., OALR
KIRKPATRICK, S.....Lauderdale Ave., Selma, Ala., OALR
KISER, J. D....Central Natl. Bk. Bldg., Bartlesville, Okla., OALR
KISTLER, O. F.....43 N. Franklin St., Wilkes-Barre, Pa., Op.AR
KISTNER, F. B.....Stevens Bldg., Portland, Ore., ALR
KITTLESON, T. N...101 Lincoln Ave., Fergus Falls, Minn., OALR
KLEENE, F.....1445 Wicker Park Ave., Chicago, Ill., OALR
KLINE, H. G.....University Blk., Syracuse, N. Y., ALR
KLINEDIENST, J. F.....220 S. George St., York, Pa., OALR
KLINGER, THOS. O.(1920)
314 Landers Bldg., Springfield, Mo., OALR
KNAPP, A.....10 E. 54th St., New York City, N. Y., Op.
*KNAPP, B.....Intermediate Life Bldg., Evansville, Ind., OALR
KNAPP, FRANK N.(1925)
203 W. Superior St., Duluth, Minn., OALR
KNEEDLER, G. C.....Jenkins Bldg., Pittsburgh, Pa.
KNIGHT, MARY S.(1925)...Mayo Clinic, Rochester, Minn., Op.
*KNODE, A. R.....Omaha Natl. Bank Bldg., Omaha, Neb., ALR
KOLLOCK, C. W.....86 Wentworth, Charleston, S. C., OALR
*KOOBS, HERMAN J. G.(1920)
4th and Walnut Sts., Rogers, Ark., OALR
*KOPETZKY, S. J....51 W. 73rd St., New York City, N. Y., ALR
*KRAFT, O. H.....25 E. Washington St., Chicago, Ill., Op.
KRAUSS, F.....1701 Chestnut St., Philadelphia, Pa., OALR
KREBS, A.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
KRENTZER, A. G..Trust Natl. Bank Bldg., Milwaukee, Wis., ALR
KRESS, G. H.....245 Bradbury Bldg., Los Angeles, Cal., OALR
*KRIEGER, G. M.....119 E. 6th St., Michigan City, Ind., OALR
KRIEGER, WM. A.(1919)
35 Market St., Poughkeepsie, N. Y., OALR
KRUG, E. F.(1908)..12 W. 44th St., New York City, N. Y., OALR

KRUSKAL, ISAAC D.(1925)
863 Prospect Pl., Brooklyn, N. Y., Op.
*KULLY, B. M.....631 City Natl. Bk. Bldg., Omaha, Neb., ALR

L

LAESSLE, HENRY A.,
The Lennox, 13th & Spruce Sts., Philadelphia, Pa., ALR
LAFORCE, B. D.....Hofman Bldg., Ottumwa, Ia., OALR
LA FORCE, E. F.....Tama Bldg., Burlington, Ia., OALR
*LAMB, F. W.(1908).....7th and Vine Sts., Cincinnati, O., OALR
LAMB, HARVEY(1920)
826 Metropolitan Bldg., St. Louis, Mo., Op.
LAMB, R. S.....Stoneleigh Ct., Washington, D. C., Op.
*LANCASTER, W. B.(1913)
520 Commonwealth Ave., Boston, Mass., Op.
LANCASTER, WILSON..310 Dakota Ave., Wahpeton, N. D., OALR
LANDES, GEORGE A.....110½ S. 18th St., Parsons, Kas., OALR
LANDFRIED, C. J.,
607 Land Title & Guarantee Bldg., New Orleans, La., ALR
LANGWORTHY, H. G.....10th & Bluff Sts., Dubuque, Ia., OALR
*LANING, J. H.(1918).726 Lathrop Bldg., Kansas City, Mo., ALR
LAPSLEY, ROBERT M.(1902).Masonic Temple, Keokuk, Ia., OALR
*LARGE, S. H.(1904)....2323 Prospect Ave., Cleveland, O., ALR
LARKIN, B. J.(1916)
Hume-Mansur Bldg., Indianapolis, Ind., Op.
LARSEN, C. L.(1916).....Lowry Bldg., St. Paul, Minn., OALR
LARUE, C. L.(1922)...17 Physicians Bldg., Boulder, Colo., OALR
LASSALLE, ALBERT.....493 St. Denis St., Montreal, Can., OALR
LASSEN, F.....First Natl. Bank Bldg., Pueblo, Colo., ALR
LA VIGNE, A. A.....11 E. 48th St., New York City, N. Y., OALR
LAWRENCE, G. A....Medical Arts Bldg., Philadelphia, Pa., Op.
LEA, JOS. A.....1125 Rialto Bldg., Kansas City, Mo., ALR
LEAVY, C. A.....Metropolitan Bldg., St. Louis, Mo., ALR
LEDERER, FRANCIS L.(1925) 2551 N. Clark St., Chicago, Ill., ALR
LEE, JOHN W.....804 Besse Bldg., Minneapolis, Minn., OALR
LEECH, J. W.....111 Broad St., Providence, R. I., OALR
*LEFLER, ANNA B.(1918)
605 W. P. Story Bldg., Los Angeles, Cal., OALR
LEHAN, J. W.....Park Pl. Bldg., Greeley, Colo., OALR
*LEMERE, H. B.....Bankers Reserve Life, Omaha, Neb., OALR
*LEMOINE, A. N.(1920)....Rialto Bldg., Kansas City, Mo., Op.
LENKER, JOHN N.....Osborn Bldg., Cleveland, O., ALR
*LENT, E. J.....122 N. Lafayette St., South Bend, Ind., OALR
LEONARD, ARTHUR C. 505 Bryant Bldg., Kansas City, Mo., OALR
LERNER, MACY L.(1923) 332 Park Ave., Rochester, N. Y., Op.
*LEVY, L.....Bank of Commerce Bldg., Memphis, Tenn., OALR
LEVY, R.....Metropolitan Bldg., Denver, Colo., ALR
*LEWIS, A. C.(1916).....Exchange Bldg., Memphis, Tenn., OALR
LEWIS, FIELDING O.....259 S. 17th St., Philadelphia, Pa., ALR
LEWIS, F. P.....454 Franklin St., Buffalo, N. Y., Op.
*LEWIS, GEORGE A.(1920)...29 Main St., Roundup, Mont., OALR

LEWIS, J. D.....516 LaSalle Bldg., Minneapolis, Minn., OALR
LEWIS, J. P.(1915).....543 Boylston St., Boston, Mass., ALR
LEWIS, WILLIAM W.(1921)
836 Lowry Bldg., St. Paul, Minn., OALR
LEWITT, F. C.(1914) 135 Stockton St., San Francisco, Cal., OAL
LIBBY, GEORGE F.(1904)....Majestic Bldg., Denver, Colo., Op.
LICHTENBERG, J. S.....Rialto Bldg., Kansas City, Mo., Op.
LIDIKAY, CHAS. J.,
519 Portsmouth Bldg., Kansas City, Kans., OALR
LIFSCHUTZ, JACOB(1925)
25 E. Washington St., Chicago, Ill.,OALR
LILLIE, HAROLD I.....Mayo Clinic, Rochester, Minn., ALR
LINDSAY, D. M.....Boston Bldg., Salt Lake City, Utah, OALR
LINN, E. G.(1913).....Fleming Bldg., Des Moines, Ia., OALR
LINN, JAY G.....820 Highland Bldg., Pittsburgh, Pa., OALR
LINTLEMAN, F. R.(1917) 312 Hayes Blk., Janesville, Wis., OALR
*LISCHKOFF, M. A.(1913)....Blount Bldg., Pensacola, Fla., OALR
LITCHFIELD, J. T.....609 Yeats Bldg., Minneapolis, Minn., OALR
LLOYD, A. W.(1915) 504 Hammond Bldg., Hammond, Ind., OALR
LLOYD, RALPH I.(1925) 14 Eighth Ave., Brooklyn, N. Y., Op.
*LOEB, C.(1907).....25 E. Washington St., Chicago, Ill., Op.
LOEB, H. W.....537 N. Grand Ave., St. Louis, Mo., ALR
LOFTUS, J. E.(1921)
605 Medical Arts Bldg., Philadelphia, Pa., ALR
LOKEY, H. M.....41 Doctors Bldg., Atlanta, Ga., OALR
LOMBARDO, MELCHIORE(1923)
186 Meserole St., Brooklyn, N. Y., Op.
*LOOMIS, E. A.....Donaldson Bldg., Minneapolis, Minn., OALR
LOOPER, EDWARD A.....605 Park Ave., Baltimore, Md., OALR
*LORIE, A.....Commerce Bldg., Kansas City, Mo., ALR
*LORING, J. BROWN.....25 E. Washington St., Chicago, Il., OALR
LOUCHERY, D. C....214 W. Main St., Clarksburg, W. Va., OALR
LOVE, JOHN G.....Ilgenfritz Bldg., Sedalia, Mo., OALR
LOWRY, BLACKBURN W.(1925)
408 Citrus Exch. Bldg., Tampa, Fla., OALR
LUBMAN, MAX....135 E. 65th St., New York City, N. Y., ALR
LUEDDE, W. H.(1916) Metropolitan Bldg., St. Louis, Mo., Op.
LUKENS, CHARLES(1906)....218 Michigan St., Toledo, O., OALR
LUKENS, ROBT. McD.,
1308 Hunting Park Ave., Philadelphia, Pa., ALR
LUX, PAUL(1925) 409 Commerce Bldg., Kansas City, Mo., ALR
*LYMAN, HARRY W.....Carleton Bldg., St. Louis, Mo., ALR
LYNCH, R. C.,
632 Maison Blanche Bldg., New Orleans, La., ALR
*LYONS, HORACE R.....30 N. Michigan Blvd., Chicago, Ill., ALR

M

MACFARLAN, D.(1916) 1805 Chestnut St., Philadelphia, Pa., OALR
*MACFARLANE, P. B...152 St. James St., S., Hamilton, Can., OALR
MACINE, J.S....503 Donaldson Bldg., Minneapolis, Minn., OALR

*MACKENZIE, ALICE V...1724 Spruce St., Philadelphia, Pa., OALR
MACKENZIE, GEORGE W.(1912)
1724 Spruce St., Philadelphia, Pa., OALR
*MAC LACHLAN, A. A.....May Bldg., Pittsburgh, Pa., ALR
MACMILLAN, JOHN A.(1925)
129 Stanley St., Montreal, Canada, Op.
MADDEN, REED.....Allen Bldg., Xenia, O., OALR
MAGEE, R. S.....Mills Bldg., Topeka, Kan., Op.A
*MAGGARD, DELANO I.....Beacon Bldg., Wichita, Kan., OALR
MAIDEN, S. D.....217 Merriam Blk., Council Bluffs, Ia., OALR
MAIRE, L. E.....Park Bldg., Detroit, Mich., Op.A
MALONEY, THOS. J.....1032 Lowry Bldg., St. Paul, Minn., OALR
MANN, R. H. T.(1913)
State Natl. Bk. Bldg., Texarkana, Tex., OALR
MANSUR, D. W.....Brockman Bldg., Los Angeles, Cal., Op.
*MARCH, CLARA A.....465 Ashland Ave., Buffalo, N. Y., Op.
MARKEL, J. C.....Westinghouse Bldg., Pittsburgh, Pa., Op.
MARLOW, SEARLE B.(1925)
University Bldg., Syracuse, N. Y., Op.
MARQUIS, G. P.....30 N. Michigan Blvd., Chicago, Ill., ALR
*MARSHALL, GEORGE G.....Gryphon Bldg., Rutland, Vt., OALR
MARSHALL, W.....Diamond Bank Bldg., Pittsburgh, Pa., ALR
MARTIN, ANCIL.....207 Goodrich Bldg., Phoenix, Ariz., Op A
MARTIN, H. H.....247 Bull St., Savannah, Ga., OALR
*MATHENY, R. C.....306 E. Main St., Galesburg, Ill., OALR
MATHESON, J.P.(1915)
511 Independence Bldg., Charlotte, N. C., OALR
MATTHEWS, J.....Metropolitan Bldg., Minneapolis, Minn., LR
MAULDIN, L. O.....Wallace Bldg., Greenville, S. C., OALR
MAUMENEE, A. E.....156 S. Francis St., Mobile, Ala., OALR
MAXEY, E. E.....4 Electric Bldg., Aberdeen, Wash., OALR
MAY, C. H.....698 Madison Ave., New York City, N. Y., Op.
MAY, J. V.....First Nat'l. Bank Bldg., Marinette, Wis., OALR
*MAY, JAMES W....800 Minnesota Ave., Kansas City, Mo., Op.
MAYBAUM, J. L.....12 E. 86th St., New York City, N. Y., ALR
MEANOR, WM. C. 619 Westinghouse Bldg., Pittsburgh, Pa., Op.
MEANS, C. S.,
1006 Pacific-Southwest Bldg., Long Beach, Cal., OALR
MEIERHOF, E. L.,
1140 Madison Ave., New York City, N. Y., OALR
*MERCER, RAY.....Stein Bldg., Quincy, Ill., OALR
*MERCER, R. E.....David Whitney Bldg., Detroit, Mich., LR
*MERRILL, H. G.....35 East, 2 South St., Provo, Utah, OALR
MERRILL, W. H.(1906)
The Bay State Bldg., Lawrence, Mass., OALR
MESSIER, A. EUGENE(1925)
303 Park Bldg., Worcester, Mass., OALR
MESSINGER, H. C.....170 Broad St., Providence, R. I., Op.
METZ, R. B.....Guardian Bldg., Cleveland, O., Op.
*METZENBAUM, M.....Rose Bldg., Cleveland, O., ALR
*MIDDLETON, A. B.....201 Sterry Blk., Pontiac, Ill., OALR

*MIDDLETON, HARRY E.....Times Bldg., Alton, Ill., OALR
MILLER, ALICE R.....West Palm Beach, Fla., OALR
MILLER, C. E.....315 S. Jefferson St., Muncie, Ind., OALR
MILLER, C. M.....Stuart Circle Hospital, Richmond, Va., OALR
MILLER, E. B.....2008 Walnut St., Philadelphia, Pa., Op.
MILLER, HUGH....520 Chambers Bldg., Kansas City, Mo., OALR
MILLETTE, J. W.....Reibold Bldg., Dayton, O., OALR
MINER, S. C.....1564 Woodward Ave., Detroit, Mich., ALR
MINOR, C. L.....Fairbanks Bldg., Springfield, O., OALR
MINOR, H. F.....Randolph Bldg., Memphis, Tenn., OALR
MINTON, WM. H.....Bartlett Bldg., St. Joseph, Mo., OALR
MITCHELL, E. K....710 W. Lehigh Ave., Philadelphia, Pa., ALR
MITHOEFER, W.....19 Garfield Pl., Cincinnati, O., ALR
MITTENDORF, A. D..399 Park Ave., New York City, N. Y., Op.
MITTENDORF, W. K.,
155 E. 53rd St., New York City, N. Y., Op.A
MOISE, A. B.....Shreveport, La., ALR
*MOLT, W. F...Newton Claypool Bldg., Indianapolis, Ind., ALR
*MONAGHAN, D. G.....535 Majestic Bldg., Denver, Colo., Op.
MONAHAN, J. A.....181 Chestnut St., Clinton, Mass., OALR
*MONOSMITH, O. B.....424 Broadway, Lorain, O., OALR
MONSON, S. H.....Ainsfield Bldg., Cleveland, O., OALR
MOORE, JOHN D.(1925)
6 Washington St., Bloomfield, N. J., OALR
MOORE, G. A.....Bank Bldg., Palmer, Mass., OALR
MOORE, GEORGE H....200 Main St., Schuylkill Haven, Pa., OALR
MOORE, M.....The Rochambeau, Washington, D. C., ALR
*MOORE, T. K.....416 Hamilton Bldg., Akron, O., OALR
*MOORE, T. W.....1050 Fifth Ave., Huntington, W. Va., OALR
MOORE, W. F.....255 S. 16th St., Philadelphia, Pa., ALR
MORGAN, J. A.....Young Bldg., Honolulu, T. Hawaii, OALR
MORRIS, A. G.....41 Gibbs St., Rochester, N. Y., OALR
MORRISON, F. A.....Willoughby Bldg., Indianapolis, Ind., Op.
MORSE, A. W.....507 Phoenix Bldg., Butte, Mont., OALR
MORSE, J. A....503 Donaldson Bldg., Minneapolis, Minn., OALR
*MORSMAN, L. W.....203 Congdon Bldg., Hibbing, Minn., OALR
MORTON, HOWARD McI.,
Metropolitan Bk. Bldg., Minneapolis, Minn., OALR
*MORTON, JOHN B.....90 E. Warren Ave., Detroit, Mich., OALR
*MOSHER, H. P.....828 Beacon St., Boston, Mass., ALR
MOSS, HOWARD S.....835 Rialto Bldg., Kansas City, Mo., OALR
MOSS, R. E.....Hicks Bldg., San Antonio, Tex., OALR
MOTT, JOHN S.....214 Argyle Bldg., Kansas City, Mo., OALR
*MOULTON, EVERETT C.,
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
MOULTON, H.,
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
*MULLIN, W. V.....Burns Bldg., Colorado Springs, Colo., ALR
*MULSOW, JOHN E.....410 Jefferson St., Milwaukee, Wis., OALR
MUNCASTER, S. B.....921, 15 A St., Washington, D. C., Op.A
MUNCY, W. M.....23 Waterman St., Providence, R. I., OALR

MUNDT, G. H.....25 E. Washington St., Chicago, Ill., OALR
MURDOCH, J. F.....Bessemer Bldg., Pittsburgh, Pa., Op.
MURPHY, J. G.....Murchison Bldg., Wilmington, N. C., OALR
MURPHY, W. E.....7th and Race Sts., Cincinnati, O., OALR
MURRAY, ALFRED N.....4654 Sheridan Rd., Chicago, Ill., OALR
*MURRAY, WM. R.....Nicollet Clinic, Minneapolis, Minn., OALR
MUSSUN, WILLIAM G.(1924) Osborn Bldg., Cleveland, O., ALR
*MYERS, ELMER A.,
407 Board of Trade Bldg., Superior, Wis., ALR
*MYERS, JOHN L.(1920)
815 Shukert Bldg., Kansas City, Mo., OALR
MYERSON, M. C.....184 Clinton St., Brooklyn, N. Y., ALR
*MCALESTER, ANDREW W., JR.,
625 Bryant Bldg., Kansas City, Mo., Op.
MCALISTER, J. C.....10 S. Mill Ave., Ridgeway, Pa., OALR
*MCBEAN, GEORGE M...22 E. Washington St., Chicago, Ill., OALR
*MCBRIDE, W. O...218 Washington Blvd., Ft. Wayne, Ind., OALR
MCCALL, JULIUS W.(1925)
515 Osborn Bldg., Cleveland, O., ALR
*MCCANNELL, ARCHIBALD D....Colcord Blk., Minot, N. D., OALR
MCCARTNEY, OLIVER P.,
314 Portsmouth Bldg., Kansas City, Kans., OALR
MCCASKEY, C. H.,
Amer. Cent. Life Bldg., Indianapolis, Ind., ALR
MCCAW, JOHN A.....418 Majestic Bldg., Denver, Colo., Op.
MCCLELLAND, L. A...78 McDonough St., Brooklyn, N. Y., ALR
*MCCLELLAND, CARL C.,
David Whitney Bldg., Detroit, Mich., OALR
MCCONNELL, T. E.....Alter Bldg., New Kensington, Pa., OALR
MCCOOL, J. L.....Stevens Bldg., Portland, Ore., Op.
*MCCORTY, VIRGIL W...800 Rialto Bldg., Kansas City, Mo., OALR
*MCCOY, J.....730 Fifth Ave., New York City, N. Y., ALR
MCCREADY, J. H.....Empire Bldg., Pittsburgh, Pa., ALR
MCCULLOUGH, R. J. P.....102 College St., Toronto, Can., OALR
*MCDERMOTT, JOHN J.(1924)
St. Joseph Sanitarium, St. Joseph, Mich., OALR
*MCDIARMID, H. O.,
Rossner Ave. and 9th St., Brandon, Manitoba, Can.,OALR
MCDONALD, C. L.....432 Ainsfield Bldg., Cleveland, O., OALR
*MCDOWELL, N. D.,...275 Alexander St., Rochester, N. Y., OALR
MCFARLANE, MURRAY....190 Bloor St., E., Toronto, Can., OALR
*MCGINNIS, EDWIN...104 S. Michigan Blvd., Chicago, Ill., ALR
MCGIVERN, CHAS. S. 805 Pacific Ave., Atlantic City, N. J., ALR
MCGUIRE, H. H....105 N. Braddock St., Winchester, Va., OALR
*MCHENRY, D. D.(1912)
604 Medical Arts Bldg., Oklahoma City, Okla., OALR
*MCKEE, GEORGE J.,
1118 Westinghouse Bldg., Pittsburgh, Pa., ALR
*MCKEE, S. H.....158 Crescent St., Montreal, Can., Op.
MCKELVEY, A. D. M.....160 Bloor St., Toronto, Can., ALR

McKennan, J. W.,
Washington Trust Bldg., Washington, Pa., Op.
McKeown, E. E.....Metropolitan Bldg., Denver, Colo., OALR
*McKinney, Alexander R.,
Bearinger Bldg., Saginaw, Mich., OALR
McKinney, R.,
Bk. of Commerce & Trust Bldg., Memphis, Tenn., ALR
McLaughlin, P. A.,
73 S. Washington St., Wilkes-Barre, Pa., OALR
*McMurray, J. B.,
Washington Trust Bldg., Washington, Pa., OALR
McNaught, H.....Butler Bldg., San Francisco, Cal., ALR
McNichol, William A.(1925)
Dixon Nat'l. Bk. Bldg., Dixon, Ill., ALR
McReynolds, G. S.....State Bank Bldg., Temple, Tex., OALR
McReynolds, J. O.....Trust Bldg., Dallas, Tex., OALR

N

Nadeau, E. G....Bellin-Buchanan Bldg., Green Bay, Wis., OALR
*Naftzger, J. B.....Davidson Bldg., Sioux City, Ia., OALR
Nance, W. O.....30 N. Michigan Blvd., Chicago, Ill., OALR
Neeper, E. R.,
Ex. Nat'l. Bank Bldg., Colorado Springs, Colo., Op.
Neighbors, Clarence A.,
Underwood Bldg., Ottawa, Kan., OALR
New, Gordon B.....Mayo Clinic, Rochester, Minn., LR
Nelson, C. F.....Schofield Bldg., Cleveland, O., OALR
*Nelson, L. A.....Lowry Arcade, St. Paul, Minn., Op.A
*Nelson, Lloyd L.....Norfolk, Neb., OALR
Newell, W. S....1029 Vermont Ave., Washington, D. C.,
*Newhart, H..... Donaldson Bldg., Minneapolis, Minn., OALR
Newton, Frank H.(1925)
907 Mercantile Bk. Bldg., Dallas, Tex., OALR
*Newton, L. A.(1919)
709 Medical Arts Bldg., Oklahoma City, Okla., OALR
Nisselson, Max 616 Madison Ave., New York City, N. Y., OALR
Noonan, Williams J.(1925)
4654 Sheridan Rd., Chicago, Ill., OALR
*Norcross, Edward P.(1924)
30 N. Michigan Ave., Chicago, Ill., ALR
Norton, Roy R.....107 B. E. State St., Sharon, Pa., OALR
*Novak, Frank, Jr.....30 N. Michigan Blvd., Chicago, Ill., ALR
Noyes, G. L.....311 Hitt St., Columbia, Mo., OALR
Nutter, C. F.....16 Amherst St., Nashua, N. H., Op.

O

O'Brien, C. S.(1924).....Colton Bldg., Toledo, Ohio, Op.
O'Brien, S. A...400 1st Nat'l. Bank Bldg., Mason City, Ia., OALR
*Oberg, C. M.....Syndicate Bldg., Minneapolis, Minn., OALR
O'Connor, D. F.....671 Broad St., Newark, N. J., OALR

O'CONNOR, R. P.....1206 Broadway, Oakland, Cal., Op.
ODELL, A.....32 Adams St., Detroit, Mich., Op.AL
O'DELL, TIMOTHY (1920)
112A Madison Ave., Aurora, Mo., OALR
OERTEL, T. E.....638 Green St., Augusta, Ga., OALR
*OESTERLING, H. E.....Winter Park, Fla., OALR
*OFFUTT, W. N.....Security Bldg., Lexington, Ky., OALR
OLSHO, SYDNEY L.....235 S. 15th St., Philadelphia, Pa., OALR
OPPENHEIMER, S....45 E. 60th St., New York City, N. Y., OALR
*ORCUTT, D. C.....7 W. Madison St., Chicago, Ill., Op.A
O'REILLY, C. A.(1911) 1901 Chestnut St., Philadelphia, Pa., ALR
O'REILLY, W. F.(1913)...44 S. Common St., Lynn, Mass., OALR
ORTON, H. B.....671 Broad St., Newark, N. J., ALR
OVERMAN, F. V...Hume-Mansur Bldg., Indianapolis, Ind., ALR
OWEN, F. S.....Brandeis Bldg., Omaha, Neb., OALR

P

PAGE, L.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
PAINTER, A. M.....Sharp Bldg., Kansas City, Mo., ALR
PALMER, EDW. M.(1919) 629 Beacon Bldg., Wichita, Kan., OALR
PARK, J. W.(1910).....32 N. Second St., Harrisburg, Pa., OALR
PARKER, E. H.....La Salle Bldg., Minneapolis, Minn., LR
*PARKER, W. R.....David Whitney Bldg., Detroit, Mich., Op.
PARRISH, MAJ. ROBERT E., M. C., U. S. A.,
Walter Reed Gen. Hospital, Washington, D. C., ALR
*PARSONS, J. G.....Lewistown, Mont., OALR
PATTERSON, E. J.....Westinghouse Bldg., Pittsburgh, Pa., ALR
*PATTERSON, J. A....Burns Bldg., Colorado Springs, Colo., OALR
PATTERSON, JOSEPH M. 518 Bryant Bldg., Kansas City, Mo., OALR
*PATTERSON, W. E. 808 La Salle Bldg., Minneapolis, Minn., OALR
*PATTON, J. M.(1910).....Brandeis Bldg., Omaha, Neb., Op.
*PAYNE, A. L.(1908)..Opera House Blk., Eau Claire, Wis., OALR
PAYNE, RICHARD J.(1925)
University Club Bldg., St. Louis, Mo., ALR
*PEARLMAN, SAMUEL J....29 E. Madison St., Chicago, Ill., ALR
*PEARSON, W. W.(1908)
Bankers Trust Bldg., Des Moines, Ia., OALR
PEELER, C. N.(1920).....W. 7th St., Charlotte, N. C., OALR
*PEIRCE, HOWARD W. David Whitney Bldg., Detroit, Mich., ALR
*PETER, LUTHER C.,
N. E. cor. Chestnut & 20th Sts., Philadelphia, Pa., Op.
PETERMAN, H. E.(1912) 512 Cathedral St., Baltimore, Md., OALR
PFAFFLIN, C. A. Newton Claypool Bldg., Indianapolis, Ind., OALR
PFINGST, A. O.....Atherton Bldg., Louisville, Ky., Op.
PFINGSTEN, C. F.....508 N. Grand Ave., St. Louis, Mo., ALR
*PHELPS, KENNETH A.(1920)
730 La Salle Bldg., Minneapolis, Minn., OALR
PHILLIPS, W. C.....40 W. 47th St., New York City, ALR
PHILLIPS, W. L.....759 Richmond Ave., Buffalo, N. Y., Op.
PHINNEY, F. D.....22 W. 7th St., Cincinnati, O., OALR
PITKIN, C. E.....Osborn Bldg., Cleveland, O., ALR

*Pollock, H. L.(1911)....2551 N. Clark St., Chicago, Ill., ALR
Pontius, P. J.....1831 Chestnut St., Philadelphia, Pa., OALR
Porter, E. H.(1912).....85 Madison St., Tiffin, O., OALR
*Porter, Jesse B.....415 S. 2nd St., Elkhart, Ind., OALR
Porter, L. B.(1913)....195 Thayer St., Providence, R. I., OALR
Posey, Wm. Campbell,
21st and Chestnut Sts., Philadelphia, Pa., Op.
*Post, Lawrence(1920)
520 Metropolitan Bldg., St. Louis, Mo., Op.
Post, Martin H.(1918)
520 Metropolitan Bldg., St. Louis, Mo., Op.
Post, Winfred B.....1380 S. Main St., Carthage, Mo., OALR
*Potter, George B.(1921)
402 City Natl. Bank Bldg., Omaha, Neb., OALR
Potter, L. F.....68 Walnut St., New Bedford, Mass., OALR
*Potter, Peter(1918)
Granate and Alaska Sts., Butte, Mont., OALR
Potts, J. B.....Brandeis Bldg., Omaha, Neb., ALR
*Powers, E.(1916)
Central Nat'l Bank Bldg., Carthage, Mo., OALR
Prangen, Avery de Hart Mayo Clinic,, Rochester, Minn., Op.
*Pratt, F. J., Jr.(1912)
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
*Pratt, J. A.(1910)
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
Prendergast, D.....1110 Euclid Ave., Cleveland, O., OALR
Price, N. W.(1908).....445 3rd St., Niagara Falls, N. Y., OALR
*Proetz, Arthur W.,
University Club Bldg., St. Louis, Mo., ALR
Proud, Willard C.(1920)
Phys. & Surg. Bldg., St. Joseph, Mo., OALR
Prout, A. W.....370 E. Town St., Columbus, O., OALR
Putnam, F. I.,
708 Sioux Falls Nat'l. Bk. Bldg., Sioux Falls, S. D., OALR
Pyle, W.(1915) 18th and Exchange Pl., Jersey City, N. J., OALR

Q

Quimby, Wm. O'G.(1919)...14 James St., Newark, N. J., OALR
Quittner, S. S.....3912 Prospect Ave., Cleveland, O., OALR

R

Radcliffe, McCluney,
1906 Chestnut St., Philadelphia, Pa., Op.
Ralls, C. T.(1916)..1st Nat'l. Bank Bldg., Winfield, Kas., OALR
*Ralston, Wallace W.(1918)
1300 Walker Ave., Houston, Tex., Op.
Ravdin, B. D.....223 Main, Evansville, Ind., OALR
*Ravdin, M.....223 Main St., Evansville, Ind., OALR
Ray, V.(1901).....19 W. 7th St., Cincinnati, O., OALR
Reaves, Chas. R. 117 W. Sycamore St., Greensboro, N. C., OALR
Reaves, W. P....117 W. Sycamore St., Greensboro, N. C., OALR

REBUCK, C. S.....412 N. Third St., Harrisburg, Pa., OALR
*RECTOR, A. E.....712 College Ave., Appleton, Wis., OALR
*REEDER, J. E.(1916).....Davidson Bldg., Sioux City, Ia., OALR
REESE, R. G.....50 W. 52nd St., New York City, N. Y., Op.
REESE, WARREN S.(1921) 230 S. 21st St., Philadelphia, Pa., Op.
REGER, H. S.....New Wellman Bldg., Jamestown, N. Y., OALR
REICHE, O. C.....328 West Broad St., Hazleton, Pa., OALR
REIM, HUGO.....417 Metropolitan Bldg., St. Louis, Mo., Op.
*REMMENS, N.....2954 Logan Blvd., Chicago, Ill., Op.
RENAUD, G. L.....Fine Arts Bldg., Detroit, Mich., OALR
RENNER, W. S.(1900)..341 Linwood Ave., Buffalo, N. Y., ALR
REPASS, ROBERT E.....150 Maple Rd., Indianapolis, Ind., ALR
*REULING, F. HAROLD(1924)....Black Bldg., Waterloo, Ia., OALR
*REYNOLDS, H. G.....Nat'l. Bank Bldg., Paducah, Ky., Op.A
*REYNOLDS, J. S.(1918)
816 La Salle Bldg., Minneapolis, Minn., OALR
*RICE, C.P.....7 Sterns Bldg., Wahpeton, N. D., OALR
RICE, G. B.(1916) 270 Commonwealth Ave., Boston, Mass., ALR
RICE JOHN E.....390 Main St., Worcester, Mass., OALR
RICE, R. H.....Coshwell Bldg., Milwaukee, Wis., Op.AL
RICHARDS, LYMAN, G.(1924)
270 Commonwealth Ave., Boston, Mass., ALR
RICHARDSON, C. W.,
1317 Connecticut Ave., Washington, D. C., ALR
RICHARDSON, J. J.,
1509 Ave. of Presidents, Washington, D. C., ALR
RICHARDSON, SHALER A.(1925)
111 W. Adams St., Jacksonville, Fla., Op.
RICHIE, L. T.(1916)...McCormick Bldg., Trinidad, Colo., OALR
*RIDEOUT, W. J.....90 Stephenson St., Freeport, Ill., OALR
RIDPATH, R. F.....1928 Chestnut St., Philadelphia, Pa., ALR
RIEGER, WALTER H.....Hanna Bldg., Cleveland, O., ALR
*RINDLAUB, ELIZABETH P.,
DeLendrecie Block, Fargo, N. D., OALR
*RINDLAUB, JOHN H.....DeLendrecie Block, Fargo, N. D., OALR
RINDLAUB, MARTIN P., JR.,
DeLendrecie Block, Fargo, N. D., OALR
*RINGLE, C. A.....Coronado Bldg., Greeley, Colo., ALR
RINGUEBERG, E. N. S.....15 Main St., Lockport, N Y., Op.
RISDON, ERNEST F.(1924) 30 Avenue Rd., Toronto, Canada, ALR
DE RIVER, JOSEPH PAUL,
U. S. Veterans' Bureau, San Francisco, Cal., OALR
*ROBERTS, S. E.(1916)
1810 Federal Reserve Bank Bldg., Kansas City, Mo., ALR
ROBERTS, W. H.....65 N. Madison Ave., Pasadena, Cal., OALR
*ROBERTSON, C. M.....30 N. Michigan, Chicago, Ill., OALR
*ROBERTSON, E. N.....132½ W. 6th St., Concordia, Kan., OALR
ROBERTSON, F. D.....Ashton Bldg., Grand Rapids, Mich., OALR
*ROBINSON, H. T.....Medical Bldg., Cumberland, Md., OALR
ROBINSON, JAMES M.....113 S. 6th St., Hiawatha, Kan., OALR
*ROBINSON, ROBERT E..205½ E. Bremer Ave., Waverly, Ia., OALR

ROGERS, A. S.....105 S. Jefferson Ave., Saginaw, Mich., OALR
ROGERS, J. T.....758 Sherbrooke St., W., Montreal, Can., ALR
ROLLER, L. A.....122 Monroe St., Grand Rapids, Mich., OALR
ROMMEL, JOHN C....4501 N. Broad St., Philadelphia, Pa., OALR
ROOKER, A. L.(1920)
1347 Michigan Ave., Niagara Falls, N. Y., ALR
ROOST, F. H.(1914).....515 Trimble Blk., Sioux City, Ia., OALR
ROSENBAUM, J.....206 Bishop St., Montreal, Can., Op.
ROTH, ALBERT W.(1920)...303 Palace Bldg., Tulsa, Okla., OALR
ROTHSCHILD, HAROLD J..239 Lowry Bldg., St. Paul, Minn., OALR
ROTT, O. M.(1915).....Paulsen Bldg., Spokane, Wash., OALR
*ROW, G. S.(1911).Hume-Mansur Bldg., Indianapolis, Ind., Op.
*ROWLAND, J. F.....Thompson Bldg., Hot Springs, Ark., OALR
ROWLAND, WILLIAM D..220 Clarendon St., Boston, Mass., Op.
RUBENDALL, C.(1915).....Brandeis Bldg., Omaha, Neb., OALR
RUBY, F. M.....Physicians Bldg., Union City, Ind., OALR
*RUCKER, E. W., JR
926 Woodward Bldg., Birmingham, Ala., ALR
*RUSH, CALVIN C.....342 Main St., Johnstown, Pa., ALR
RUSSELL, EDWIN L.(1920)
402 Argyle Bldg., Kansas City, Mo., OALR
*RUSSELL, J. A.....206 Masonic Temple, Erie, Pa., ALR
RUSSELL, RALPH E.....Waterloo, Iowa, OALR
RUST, E. G.....Hanna Bldg., Cleveland, O., Op.A
RUTHERFORD, CYRUS W.(1925)
Pennway Bldg., Indianapolis, Ind., Op.
RYAN, LAWRENCE R.,
San Marcos Bldg., Santa Barbara, Cal., Op.A
RYDER, D. R.....151 Rock St., Fall River, Mass., ALR
RYERSON, FRANK L.(1925)
307 Fine Arts Bldg., Detroit, Mich., OALR

S

*SACCO, ANTHONY G.(1920)
440 Clinton Ave., West Hoboken, N. J., OALR
*SALINGER, SAMUEL(1920)
25 E. Washington St., Chicago, Ill., ALR
SALLANDER, FREDERICK W.(1919)
303 Security Bank Bldg., Sioux City, Ia., OALR
*SANDERSON, D. D.(1917).....Funke Bldg., Lincoln, Neb., OALR
*SANDERSON, H. H...David Whitney Bldg., Detroit, Mich., OALR
SARTAIN, PAUL J.(1921)
2006 Walnut St., Philadelphia, Pa., Op.
SATTERLEE, R. H.....187 Delaware Ave., Buffalo, N. Y., Op.
SAUER, W. E.....Humboldt Bldg., St. Louis, Mo., ALR
SAUER, W. W.(1910).....Box 416, Marietta, O., OALR
SAVERY, C. E.....126 N. Lafayette St., South Bend, Ind., OALR
SCALES, H. L.(1920)
504-5 Rorabaugh-Wiley Bldg., Hutchinson, Kas., OALR
SCALES, J. L.....Commercial Bank Bldg., Shreveport, La., OALR
SCALES, J. W.....202½ Main St., Pine Bluff, Ark., OALR

SCARLET, HUNTER W. (1919)
2228 Locust St., Philadelphia, Pa., Op.
SCHAFFERT, N. L. . 57 S. Washington St., Wilkes-Barre, Pa., OALR
SCHATZ, HENRY A. . . . 2035 Chestnut St., Philadelphia, Pa., ALR
SCHENCK, CHARLES P. (1920)
604-7 Ft. Worth Natl. Bank Bldg., Fort Worth, Tex., OALR
*SCHILD, E. H. (1906)
Cleveland Ave. and 9th St., Canton, O., Op.A
SCHILLER, A. NOAH,
225 West End Ave., New York City, N. Y., ALR
SCHLANSER, ADAM E. (1921) . . . . . Ft. Leavenworth, Kas., OALR
SCHLEGEL, H. T. . . . . 501 Third St., Wausau, Wis., OALR
*SCHLINDWEIN, G. W. . . . . 138 W. 9th St., Erie, Pa., OALR
SCHLIVEK, KAUFMAN (1925)
1000 Park Ave., New York City, N. Y., Op.
SCHNEIDEMANN, T. B
Professional Bldg., Philadelphia, Pa., Op.
SCHOLZ, R. P. . . . . Metropolitan Bldg., St. Louis., Mo., ALR
SCHRANT, J. H. . . 504 Natl. Bank Bldg., Hutchinson, Kan., OALR
SCHURTER, M. A. . . . . 8 Morton Ave., Riverside, N. J., OALR
*SCHUSTER, STEPHEN A. . 1st Natl. Bk. Bldg., El Paso, Tex., ALR
SCHUTZ, W. H. . . . . 712 Bryant Bldg., Kansas City, Mo., OALR
SCHWARTZ, FREDERICK A. (1919)
601 Metropolitan Bldg., St. Louis, Mo., Op.
SCHWEINITZ, GEO. E. DE,
1705 Walnut St., Philadelphia, Pa., Op.
SCHWENK, P. N. K. . . 1417 N. Broad St., Philadelphia, Pa., Op.
*SCOTT, JOHN R. (1920) . . . . . 201 S. Main St., Ottawa, Kan., OALR
*SCROGGS, J. J. . . . . 218 E. Third St., Beaver, Pa., OALR
SEAMAN, GILBERT E.,
309 Goldsmith Bldg., Milwaukee, Wis., OALR
*SEARCY, H. B. . . . . 807 Greensboro Ave., Tuscaloosa, Ala., OALR
SEARS, WM. H. (1910) . . . . . 514 Penn St., Huntingdon, Pa., OALR
SEAY, D. E. . . . . Dallas Co. State Bk. Bldg., Dallas, Tex., OALR
*SECOY, FRANK L. (1922) . 525 Francis Bldg., Sioux City, Ia., OALR
SEDGWICK, O. W. (1917) . . . . . Box 494, Ionia, Mich., OALR
SEDWICK, W. A. . . . . Imperial Bldg., Denver, Colo., OALR
*SEEGMAN, S. . . . . Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
SEELYE, WALTER K. (1920)
806 American Bank Bldg., Seattle, Wash., OALR
SELFRIDGE, G. L. . . . . Schroth Bldg., San Francisco, Cal., ALR
SELLERS, LYLE M. . . . . 430 Argyle Bldg., Kansas City, Mo., ALR
SENER, WALTER J. . . . . 130 S. 18th St., Philadelphia, Pa., OALR
SERNOFFSKY, ISAAC. . . . . 37 Allen St., Buffalo, N. Y., Op.
SEWALL, E. C. . . . . Lane Hospital, San Francisco, Cal., OALR
*SEYDELL, E. M. (1916) . . . . . 201 N. Main St., Wichita, Kas., ALR
SHACKLETON, W. E. (1918)
2323 Prospect Ave., Cleveland, O., Op.
SHAFER, J. J. (1913) . . . . . Stark Bldg., Louisville, Ky., OALR
SHAHAN, W. E. . . . . Metropolitan Bldg., St. Louis, Mo., Op.
SHANAHAN, T. J. (1914) . . 419 Boylston St., Boston, Mass., ALR

SHANKLIN, E. M.....575 Hohman St., Hammond, Ind., OALR
SHANNON, C. E.....104 Copley Rd., Upper Darby, Pa., Op.
SHAPERO, I. M.(1919)....365 East Ave., Rochester, N. Y., OALR
SHARRETT, GEORGE O.(1921)
119 Bedford St., Cumberland, Md., OALR
*SHASTID, T. H.(1913)...400 Lyceum Bldg., Duluth, Minn., Op.
SHAWHAN, GLENN E.....Nampa, Ida., OALR
*SHEA, J. J.(1916).....Exchange Bldg., Memphis, Tenn., OALR
SHELLMAN, J. L.(1917)....Lowry Bldg., St. Paul, Minn., OALR
*SHEMELEY, WM. G., JR..1724 Spruce St., Philadelphia, Pa., OALR
SHERMAN, MAJ. A. EDWARD
Station Hospital, Ft. Sam Houston, Tex., OALR
*SHERMAN, E. S.(1912).....Wiss Bldg., Newark, N. J., OALR
*SHIELDS, J. M.....Metropolitan Bldg., Denver, Colo., Op.
SHIELDS, LAWRENCE.....Allen Bldg., Xenia, O., ALR
SHIRAS, HOWARD H....629 Guardian Bldg., Cleveland, O., Op.
SHIREY, OSCAR M.(1925).....Osborn Bldg., Cleveland, O., ALR
SHOEMAKER, FERDINAND....728 Union St., Emporia, Kas., OALR
SHOEMAKER, S. A., U. S. Veterans' Hospital No. 67,
11th and Harrison Sts., Kansas City, Mo., OALR
SHOEMAKER, W. A.....Carleton Bldg., St. Louis, Mo., Op.
SHORT, Z. N.....Dugan Stuart Bldg., Hot Springs, Ark., OALR
SHREVE, O. M.(1905).....162 W. 8th St., Erie, Pa., OALR
SHULTS, EDWIN J. G.(1920).Haines Bldg., Eldorado, Kas., OALR
SHUMAN, G. H.(1917)..1105 Park Bldg., Pittsburgh, Pa., Op.
*SHUMATE, DAVID L..517 Shukert Bldg., Kansas City, Mo., OALR
*SHURLY, B. R.....62 W. Adams St., Detroit, Mich., ALR
SHUTE, D. K.....1717 DeSales St., Washington, D. C., Op.
SIEGEL, F. X....2700 Union Central Bldg., Cincinnati, O., OALR
*SILBERNAGEL, C. E.....283 E. State St., Columbus, O., OALR
SIMONDS, O. F.....Hanna Bldg., Cleveland, O., Op.
*SIMPSON, H. LEE,
1413 David Whitney Bldg., Detroit, Mich., ALR
*SIMPSON, JOHN R.,
1005 Westinghouse Bldg., Pittsburgh, Pa., ALR
SIMPSON, W. L.....Exchange Bldg., Memphis, Tenn., OALR
SINGLETON, E. M.(1897)
111 E. Church St., Marshalltown, Ia., OALR
*SKILLERN, R. H.(1906)
1928 Chestnut St., Philadelphia, Pa., LR
SKILLERN, SAMUEL R. JR.(1925)
2008 Walnut St., Philadelphia, Pa., ALR
SLEIGHT, R. D.(1906).....Post Bldg., Battle Creek, Mich., OALR
SLOAN, HENRY L.(1919)
Independence Bldg., Charlotte, N. C., Op.
*SLOCUM, G.....328 E. Huron St., Ann Arbor, Mich., Op.
SLUDER, G.....3542 Washington Ave., St. Louis, Mo., ALR
SMITH, ARTHUR E.,
1016 Donaldson Bldg., Minneapolis, Minn., OALR
SMITH, D.....834 Myrtle Ave., Bridgeport, Conn., Op.A
*SMITH, D. S.(1917)....State Bank Bldg., La Crosse, Wis., OALR

SMITH, E. T....Connecticut Mut. Bldg., Hartford, Conn., Op.
*SMITH, G. B.(1914) Broad St. and Fourth Ave., Rome, Ga., OALR
*SMITH, H. A.....640 Main St., Delta, Colo., OALR
SMITH, J. M.....134½ N. Patterson St., Valdosta, Ga., OALR
SMITH, R. C.....Board of Trade Bldg., Superior, Wis., OALR
SMITH, S. McC.....1420 Spruce St., Philadelphia, Pa., ALR
SMITH, S. S.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
SMITH, V. C.(1912) 722 Audubon Bldg., New Orleans, La., Op.
SNEED, CARL M.....506 W. Broadway, Columbia, Mo., OALR
SNELL, A. C.(1925)..53 S. Fitzhugh St., Rochester, N. Y., Op.
SNOW, L. W.....Deseret Bank Bldg., Salt Lake City, Utah, OALR
*SNYDER, W. H.....211 Ontario St., Toledo, O., OALR
SOMBERG, JOSEPH S.....45 E. 60th St., New York, N. Y., Op.
*SONNENSCHEIN, R.....Heyworth Bldg., Chicago, Ill., ALR
*SPAKE, L. B.(1920)
522 Minnesota Ave., Kansas City, Kans., ALR
SPALDING, J. A.(1896)....627 Congress St., Portland, Me., Op.A
*SPENCER, F. R.(1904)...Physicians Bldg., Boulder, Colo., OALR
SPENCER, W....1830 S. Rittenhouse Sq., Philadelphia, Pa., OALR
SPENGLER, J. A.....423 Main St., Geneva, N. Y., OALR
*SPITZE, E. C.(1916)....Murphy Bldg., East St. Louis, Ill., Op.
STAHLMAN, F. C.....318 Fifth St., C[illegible]leroi, Pa., OALR
STANBERRY, H.....Doctors Bldg., Cincinnati, O., Op.AR
*STANFORD, J. B.....Exchange Bldg., Memphis, Tenn., OALR
STARK, JESSE B.(1925)..270 Park Ave., New York, N. Y., Op.
STARR, E. G.....523 Delaware Ave., Buffalo, N. Y., Op.
STAUFFER, F.....Deseret Bk. Bldg., Salt Lake City, Utah, OALR
*STEGMAN, L. B.....Sanitarium, Battle Creek, Mich., Op.
STEIM, J, M...First Natl. Bk. Bldg., New Kensington, Pa., Op.
STEIN, JULES C.(1924) 22 E. Washington St., Chicago, Ill., Op.
*STEIN, O. J.....30 N. Michigan Blvd., Chicago, Ill., ALR
STEINFELD, A. L.(1922).....234 Michigan St., Toledo, O., OALR
STELLE, HARRY L...207 Commerce Bldg., Pittsburg, Kan., OALR
*STEVENSON, W. D.....165 E. Market St., Akron, O., OALR
*STEVENSON, W. D.....Majestic Bldg., Quincy, Ill., OALR
STEWART, C. S.....106 N. Main St., Auburn, Ind., OALR
*STEWART, WALTER E....Tribune Bldg., Terre Haute, Ind., OALR
STICKNEY, OTIS D.(1920)
92 Pacific Ave., Atlantic City, N. J., ALR
STIEREN, E.(1905) 626 Union Trust Bldg., Pittsburgh, Pa., Op.
*STILES, C. M.....1831 Chestnut St., Philadelphia, Pa., Op.
STILLSON, H.....Seaboard Bldg., Seattle, Wash., OALR
STIMSON, GEORGE W.(1919)
Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
STOCKLER, J. A.....332 North Fifth St., Reading, Pa., OALR
STOTTER, ARTHUR L.....1148 Euclid Ave., Cleveland, O., OALR
STOTTER, J.....1148 Euclid Ave., Cleveland, O., OALR
STOUT, PHILIP S....Medical Arts Bldg., Philadelphia, Pa., ALR
STRADER, G. L.(1904)..408 Hynds Bldg., Cheyenne, Wyo., OALR
STRAUSS, JEROME F.(1925)
104 S. Michigan Blvd., Chicago, Ill., ALR

STRICKER, L. . . . . The Groton, Cincinnati, O., Op.
STRICKLER, D. A. . . . . 615 Empire Bldg., Denver, Colo., OALR
STRONG, J. C. . American Natl. Bank Bldg., Leadville, Colo., OALR
STROUSE, FRED. M. . . . . 1301 Spruce St., Philadelphia, Pa., ALR
*STROUT, E. S. (1918) . . . 74 S. 11th St., Minneapolis, Minn., OALR
STROUT, G. ELMER. . . Donaldson Bldg., Minneapolis, Minn., OALR
STUCKY, J. A. (1896)
311 Guaranty Bank Bldg., Lexington, Ky., OALR
*STUCKY, W. S. . 311 Guaranty Bank Bldg., Lexington, Ky., OALR
STUEBER, F. G. (1905) . . . . . Main and North Sts., Lima, O., OALR
STUEBER, P. J. (1917) . . . . . 209 W. North St., Lima, O., OALR
STURGIS, LEIGH F. (1920)
128 W. 87th St., New York City, N. Y., ALR
STURM, S. A. . . . . Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*SUKER, G. F. . . . . 25 E. Washington St., Chicago, Ill., Op.
SULLIVAN, JOHN J., JR. . Traders Bank Bldg., Scranton, Pa., ALR
SULZER, G. A. . . . . 200 E. State St., Columbus, O., OALR
SULZMAN, F. M. (1922) . . . . . 1831 Fifth Ave., Troy, N. Y., OALR
*SWAN, C. J. (1911) . . . . . The Gables Bldg., Evanston, Ill., OALR
SWEET, W. M. . . . . 1205 Spruce St., Philadelphia, Pa., Op.
SWETNAM, C. R. K. (1915) Masonic Temple, Prescott, Ariz., ALR

T

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607 Union Central Bldg., Cincinnati, O., ALR
TAQUINIO, GEORGE J.,
Maison Blanche Bldg., New Orleans, La., ALR
TARUN, W. . . . . 605 Park Ave., Baltimore, Md., Op.A
*TAYLOR, CHAS. B. (1921) . . . . . Ennis Bldg., Ottumwa, Ia., ALR
TAYLOR, L. E. (1905)
Marshall Field Annex Bldg., Chicago, Ill., ALR
TAYLOR, LEWIS H. . . 83 S. Franklin St., Wilkes-Barre, Pa., OALR
*TAYLOR, M. C. . . . . 415 Warrington Ave., Pittsburgh, Pa. Op.
THIGPEN, C. A. . First Natl. Bank Bldg., Montgomery, Ala., OALR
*THOMAS, CHAS. D. . . Central Natl. Bank Bldg., Peoria, Ill., OALR
*THOMAS, FRANCES W. (1916) . . 74 S. 5th St., Columbus, O., OALR
THOMAS, H. G. . . . . Dalziel Bldg., Oakland, Cal., OALR
*THOMASON, H. E. . . . . Rialto Bldg., Oakland, Cal., OALR
THOMASSON, W. J. . . . . 942 York St., Newport, Ky., OALR
THOMPSON, E. H.,
Vindonissa Bldg., 19 W. 7th St., Cincinnati, O., OALR
*THOMPSON, G. H. (1909)
18 Ashland St., North Adams, Mass., OALR
THOMPSON, H. M. (1912)
First Natl. Bank Bldg., Pueblo, Colo., Op.
THOMPSON, J. J. . . . . 3 Park Ave., Mt. Vernon, N. Y., OALR
THOMPSON, P. H.,
308 Commonwealth Ave., Boston, Mass., Op.
*TILDERQUIST, D. L. . . . . 7 E. Superior St., Duluth, Minn., OALR
TIMBERMAN, A. . . . . City Bank Bldg., Columbus, O., OALR
TINDOLPH, L. W. . . . . 1st Natl. Bank Bldg., Olean, N. Y., OALR

TINGLEY, LOUISA PAINE(1913)
9 Massachusetts Ave., Boston, Mass., Op.
TITSWORTH, GUY.....111 W. 4th St., Sedalia, Mo., Op.
*TIVNEN, R. J.....104 S. Michigan Blvd., Chicago, Ill., OALR
*TOBEY, G. L.....270 Commonwealth Ave., Boston, Mass., ALR
*TODD, G. C.(1922)
307 Westinghouse Bldg., Pittsburgh, Pa., OALR
TODD, H. COULTER...Colcord Bldg., Oklahoma City, Okla., OALR
*TOMASSENE, RAYMOND A.,
Wheeling Steel Corp. Bldg., Wheeling, W. Va., OALR
*TOMLIN, W. S....Hume-Mansur Bldg., Indianapolis, Ind., ALR
TOMS, S. W. S.(1909).....Nyack-on-Hudson, N. Y., ALR
*TOOKER, C. W.(1907).....Carleton Bldg., St. Louis, Mo., Op.
TOWNSEND, JOHN F.(1923)
85 Wentworth, Cor. St. Phillips St., Charleston, S. C., OALR
TRIBLE, GEORGE B.,
1026 16th St., N. W., Washington, D. C., OALR
TRIMBLE, CLARENCE S.(1920)
705 Commercial St., Emporia, Kan., OALR
TRIPP, IRA C.(1920).....436 Rose Bldg., Cleveland, O., OALR
TRIPP, LEROY R.....307 Security Bldg., Sioux City, Iowa, OALR
*TUCKER, GABRIEL F.....335 Trevor Lane, Cynwyd, Pa., ALR
TUCKER, JAMES C.(1920).....Beatrice, Neb., OALR
*TUCKERMAN, W. C.(1911)...Osborn Bldg., Cleveland, O., Op.
*TUCKERMAN, W. H.....Osborn Bldg., Cleveland, O., OALR
TUREMAN, HERBERT G.,
1100 Rialto Bldg., Kansas City, Mo., OALR
TURNBULL, F. M.(1916)
504 Providence Bldg., Duluth, Minn., ALR
TURNER, H. H.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
TURNER, O. W.....Augusta Trust Bldg., Augusta, Me., OALR
TWITTY, W. C...Peoples Natl. Bk. Bldg., Rock Hills, S. C., OALR

U

UPHAM, H. F.....305 Third Ave., Asbury Park, N. J., OALR
*UREN, CLAUDE THOMAS
325 Omaha Natl. Bank Bldg., Omaha, Neb., OALR
URNER, M. H....Union Central Life Bldg., Cincinnati, O., OALR

V

*VAIL, D. T.....24 E. 8th St., Cincinnati, O., Op.
*VAIL, H. H.(1924).....24 E. 8th St., Cincinnati, O., ALR
*VAN KIRK, FRANK J.,
Bell. Natl. Bank Bldg., Bellingham, Wash., OALR
VANKIRK, V. E.....Union Arcade Bldg., Pittsburgh, Pa., Op.
VAN POOLE, GIDEON McD.,
Young Bldg., Honolulu, T. Hawaii, OALR
*VAUGHAN, G. E.....711 S. 3rd St., Louisville, Ky., OALR
VAUGHN, EDGAR H...113½ W. Ferguson St., Tyler, Tex., OALR
VEASEY, C. A.....Paulsen Bldg., Spokane, Wash., OALR

VINSONHALER, F.....Urquhart Bldg., Little Rock, Ark., OALR
VOIGHT, C. B.....1702 Broadway, Mattoon, Ill., OALR
*VON COLDITZ, G. F....25 E. Washington St., Chicago, Ill., OALR
VOORHEES, I. W
14 Central Park, W., New York City, N. Y., ALR

W

WAGERS, ARTHUR J.,
The Lenox, 13th and Spruce Sts., Philadelphia, Pa., ALR
*WAGNER, C. B.....31 North State St., Chicago, Ill., OALR
WALES, E. DEWOLF...1236 N. Penn St., Indianapolis, Ind., ALR
WALKER, CHAS. C..Woodward at Warren, Detroit, Mich., Op.
*WALKER, C. E.....Jacobson Bldg., Denver, Colo., Op.
*WALKER, H. S.....77 E. Washington St., Chicago, Ill., OALR
WALLACE, F. E.....517 Thatcher Bldg., Pueblo, Colo., OALR
WALSH, JOSEPH M.....Rapid City, S. D., OALR
*WALTER, WILL.....1414 Chicago Ave., Evanston, Ill., OALR
WATSON, H. D.(1915)..151 Front St., Binghampton, N. Y., OALR
WATSON, JAMES A.,
500 Phys. and Surg. Bldg., Minneapolis, Minn., OALR
*WATSON, R. SEYMOUR,
120 N. Michigan Ave., Saginaw, Mich., OALR
*WAUGH, J. M.,
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., LR
WEAVER, T. W.....201 N. Main St., Wichita, Kan., Op.
*WEED, H. M.....196 Lincoln Ave., Buffalo, N. Y., Op.
WEEKS, JOHN H....20 E. 53rd St., New York City, N. Y., Op.
WEEKS, W. W.....20 E. 53rd St., New York City, N. Y., Op.
*WEIH, ELMER PAUL.....605 Wilson Bldg., Clinton, Iowa, OALR
*WEIL, A. L..717 Maison Blanche Annex, New Orleans, La., ALR
WEIMER, E. S.....332 Lehigh Ave., Pittsburgh, Pa., Op.
WEISSER, E. A.....Empire Bldg., Pittsburgh, Pa., Op.
WELCH, R. S. G.,
League Island Nav. Hosp., Philadelphia, Pa., OALR
WELLS, D. W.....Hotel Westminster, Boston, Mass., Op.
WELLS, G. S.....San Marcos Bldg., Santa Barbara, Cal., ALR
WELLS, W. A.....The Rochambeau, Washington, D. C., OALR
WELSH, D. E.(1896)
Powers Theater Bldg., Grand Rapids, Mich., OALR
*WELTY, C. F.....Shreve Bldg., San Francisco, Cal., ALR
*WENCKE, C. G..16 Wentworth Court, Battle Creek, Mich., OALR
*WENDEL, JACOB S....David Whitney Bldg., Detroit, Mich., ALR
WESTCOTT, C. D.....22 E. Washington, Chicago, Ill., Op.
WESTLAKE, S. V.....Humboldt Bldg., St. Louis, Mo., ALR
WHALEY, E. M.....1430 Blanding Bldg., Columbia, S. C., OALR
WHEELER, ERNEST CALTRON,
Tacoma Clinic, Tacoma, Wash., OALR
*WHEELER, JOHN M., 30 W. 59th St., New York City, N. Y., Op.
WHEELER, MERRITT W.....Lowry Bldg., St. Paul, Minn., OALR
WHELAN, GEORGE L.(1925)
257 S. 16th St., Philadelphia, Pa., ALR

*WHERRY, W. P..620 Bankers Reserve Bldg., Omaha, Neb., OALR
WHISNANT, A. M.....Realty Bldg., Charlotte, N. C., OALR
*WHITAKER, J.....Hume-Mansur Bldg., Indianapolis, Ind., OALR
WHITE, C. P.....2327 W. 18th St., Wilmington, Del., OALR
WHITE, E. H.....589 Dorchester St., Montreal, Can., ALR
WHITE, L. E.(1912)
390 Commonwealth Ave., Boston, Mass., ALR
WIBLE, E. E.....Diamond Bank Bldg., Pittsburgh, Pa., Op.
WIENER, A.(1909)..550 Park Ave., New York City, N. Y., OALR
*WIENER, M.....Carleton Bldg., St. Louis, Mo., Op.
*WILBUR, E. P.,
Kalamazoo Natl. Bank Bldg., Kalamazoo, Mich., OALR
*WILDER, W. H.....122 S. Michigan Blvd., Chicago, Ill., Op.
WILLARD, L. M.....520, 3rd St., Wausau, Wis., OALR
WILLIAMS, HORACE J.(1922)
5908 Green St., Germantown, Philadelphia, Pa., ALR
WILLIAMS, J. P.....Funke Bldg., Lincoln, Neb., OALR
*WILLIAMS, THOS. J...30 N. Michigan Blvd., Chicago, Ill., OALR
*WILLIAMS, W. C.....Jefferson Bldg., Peoria, Ill., OALR
*WILLIAMS, WALTER H.....Middletown, O., OALR
WILMER, FRANCIS M.....Castell Bldg., Winfield, Kan., OALR
*WINTER, G. E.(1921).....44 Sun Bldg., Jackson, Mich., OALR
*WINTER, JOHN ARTHUR.....Fidelity Bldg., Duluth, Minn., OALR
*WISE, RALPH C.....News Bldg., Mansfield, O., OALR
WOLD, KARL C.(1924)
7th and Wacouta Sts., St. Paul, Minn., OALR
WOLFE, A. C.....350 E. State St., Columbus, O., ALR
*WOLFE, C. T.....Francis Bldg., Louisville, Ky., OALR
*WOLFE, OTIS R.(1919)
908 W. Boone St., Marshalltown, Iowa, OALR
WOLFENSTEIN, LEO.....746 Rose Bldg., Cleveland, O., Op.
WOOD, C. A.....7 W. Madison St., Chicago, Ill., Op.
WOOD, D.....The Donaldson Bldg., Minneapolis, Minn., OALR
*WOOD, HILLIARD.....Independent Bldg., Nashville, Tenn., OALR
WOODRUFF, F. E.....Metropolitan Bldg., St. Louis, Mo., Op.
*WOODRUFF, H. W.....305 Heggie Bldg., Joliet, Ill., OALR
*WOODRUFF, T. A.....Plant Bldg., New London, Conn., Op.
WOODS, HIRAM.....842 Park Ave., Baltimore, Md., Op.A
*WOODS, R. H.....Neustadt Bldg., La Salle, Ill., OALR
*WOODSON, J. M.....City Natl. Bank Bldg., Temple, Tex., OALR
*WOODWARD, J. M.....Richards Blk., Lincoln, Neb.
WORMLEY, H. R.....410 Brown Bldg., Rockford, Ill., OALR
WRIGHT, CHAS. D.(1921)
1111 Metropolitan Bank Bldg., Minneapolis, Minn., OA
WRIGHT, J. B....Citizens Natl. Bank Bldg., Raleigh, N. C., OALR
WRIGHT, J. W....Central Natl. Bank Bldg., Columbus, O., Op.
*WRIGHT, J. W. M.,
Hume-Mansur Bldg., Indianapolis, Ind., ALR
WRIGHT, K. L.....247 W. 8th St., Erie, Pa., ALR
*WURTZ, W. J. M....2808 Delaware Ave., Kenmore, N. Y., ALR

WÜRDEMANN, H. V.....Cobb Bldg., Seattle, Wash., OALR
*WYLIE, C. B.....293 High St., Morgantown, W. Va., OALR

Y

YANKAUER, S...616 Madison Ave., New York City, N. Y., ALR
YAZEL, HERMAN E.,
510-511 Grand Ave. Temple, Kansas City, Mo., OALR
*YOUNG, H. B.....204 Jefferson St., Burlington, Iowa, OALR
YOUNG, H. H.....Morrison Bldg., Charleston, W. Va., OALR

Z

ZEHNDER, A. CHARLES (1919)
180 Fairmount Ave., Newark, N. J., Op.
*ZEMER, S.....Security Mutual Bldg., Lincoln, Neb., OALR
*ZENTMAYER, WILLIAM..1506 Spruce St., Philadelphia, Pa., Op.
ZINKHAN, ARTHUR M.,
1025 Fifteenth St., N. W., Washington, D. C., ALR

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ALABAMA

BIRMINGHAM

HARRISON, W. G. ..... Empire Bldg., OALR
RUCKER, EDMOND W. J. ..... Woodward Bldg., ALR

MOBILE

MAUMENEE, A. E. ..... 156 St. Francis St., OALR

MONTGOMERY

THIGPEN, C. A. ..... First National Bank Bldg., OALR

SELMA

KIRKPATRICK, SAMUEL ..... Lauerdale Ave., OALR

TUSCALOOSA

SEARCY, H. B. ..... 807 Greensboro Ave., OALR

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MARTIN, ANCIL ..... 207 Goodrich Bldg., Op.A

PRESCOTT

SWETNAM, C. R. K. ..... Masonic Temple, ALR

ARKANSAS

EUREKA SPRINGS

HUNTINGTON, ROBT. H. ..... OALR

FORT SMITH

BUCKLEY, J. H. ..... 1st National Bank Bldg., OALR
DORENTO, DRED R. ..... Merchants National Bank Bldg., OALR
MOULTON, EVERETT C. ..... Merchants National Bank Bldg., OALR
MOULTON, HERBERT ..... Merchants National Bank Bldg., OALR

HOT SPRINGS

KING, O. H. ..... Dugan-Stuart Bldg., OALR
ROWLAND, J. F. ..... Thompson Bldg., OALR
SHORT, Z. N. ..... Dugan-Stuart Bldg., OALR

LITTLE ROCK

CALDWELL, ROBERT ..... State National Bank Bldg., OALR
VINSONHALER, FRANK ..... Urquhart Bldg., OALR

PINE BLUFF

CRUMP, J. F. ..... Citizens' Bank Bldg., OALR
SCALES, J. W. ..... 202½ Main St., OALR

ROGERS

KOOBS, H. J. G. ..... 4th and Walnut Sts., OALR

CALIFORNIA

HOLLYWOOD

KERRICK, STANLEY E.....Security Bldg., OALR

LONG BEACH

MEANS, C. S.....Pacific-Southwest Bldg., OALR

LOS ANGELES

BELLIN, JOSEPH.....1026 W. 6th St., OALR
BOURBON, OLIVER P.....222 Westlake Professional Bldg., OALR
BROWN, J. MACKENZIE.....1136 W. 6th St., ALR
DETLING, F.....Investment Bldg., OALR
DUDLEY, WM. H.....Brockman Bldg., OALR
FISHER, CARL.....1028 Pacific Mut. Bldg., Op.A
GOSNEY, C. W.....6075 Franklin Ave., OALR
IRVINE, A. R.....709 Westlake Professional Bldg., Op.
KIEFER, HUGO A.....Brockman Bldg., OALR
KRESS, GEO. H.....Bradbury Bldg., OALR
LEFFLER, ANNA B.....W. P. Story Bldg., OALR
MANSUR, D. W.....Brockman Bldg., Op.

OAKLAND

O'CONNOR, R. P.....1206 Broadway, Op.
THOMAS, H. C.....Dalziel Bldg., OALR

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BROWN, H. A.....719 St. Paul Bldg., Op.
DOLMAN, P.....Flood Bldg., Op.
GRAHAM, H. B.....210 Post St., ALR
GREEN, AARON S.....853 Hyde St., Op.
GREEN, LOUIS D.....853 Hyde St., Op.
ISRAEL, J. P.....U. S. Veterans' Bureau, OALR
LEWITT, F. C.....135 Stockton St., OAL
MCNAUGHT, H.....Butler Bldg., ALR
DERIVER, JOSEPH.....U. S. Veterans' Bureau, OALR
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BORDLEY, JAMES, JR.....330 N. Charles St., Op.
FRIEDENWALD, HARRY.....1029 Madison Ave., Op.A
FRIEDENWALD, JONAS S.....1212 Eutaw Pl., Op.
GOLDBACH, L. J.....1012 N. Charles St., OALR
KEMLER, JOSEPH J.....1908 Eutaw Pl., OALR
LOOPER, EDWARD A.....605 Park Ave., OALR
PETERMAN, H. F.....512 Cathedral St., OALR
TARUN, WILLIAM.....605 Park Ave., Op.A
WODDS, HIRAM.....842 Park Ave., Op.A

CUMBERLAND

JONES, E. L.....First National Bank Bldg., OALR
ROBINSON, H. T.....Medical Bldg., OALR
SHARRETT, GEORGE O.....119 Bedford St., OALR

MASSACHUSETTS

BOSTON

BRYANT, A. G.....502 Beacon St., ALR
CARVILL, L. MAUD.....82 Commonwealth Ave., Op.

CHENERY, W. E.....377 Commonwealth Ave., ALR
CORBETT, JEREMIAH J.....520 Beacon St., OALR
DERBY, G. S.....23 Bay State Road, Op.
FREEDMAN, L. M.....419 Boylston St., ALR
GREENWOOD, ALLEN.....82 Commonwealth Ave., Op.
HURLEY, E. D.....419 Boylston St., Op.
HURLEY, E. H.....419 Boylston St., Op.
INGLIS, H. J.....Bay State Bldg., ALR
LANCASTER, W. B.....529 Commonwealth Ave., Op.
LEWIS, J. P.....543 Boylston St., ALR
MOSHER, H. P.....828 Beacon St., ALR
RICE, G. B.....293 Commonwealth Ave., ALR
RICHARDS, LYMAN G.....270 Commonwealth Ave., ALR
ROWLAND, WILLIAM D.....220 Clarendon St., Op.
SHANAHAN, T. J.....419 Boylston St., ALR
STANDISH, MYLES.....51 Hereford St., Back Bay., Op.
THOMPSON, P. H.....308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE.....9 Massachusetts Ave., Op.
TOBEY, G. L.....270 Commonwealth Ave., ALR
WELLS, D. W.....The Westminster, Op.
WHITE, L. E.....390 Commonwealth Ave., ALR

CLINTON

MONAHAN, J. A.....181 Chestnut St., OALR

FALL RIVER

RYDER, D. R.....151 Rock St., ALR

FRAMINGHAM

JESSAMAN, L. W.....182 Concord St., OALR

GARDNER

BONE, H. D.....29 Parker St., OALR

HOLYOKE

HUSSEY, E. J.....276 High St., Op.LR

LAWRENCE

CONLON, F. A.....Bay State Bldg., OALR
MERRILL, W. H.....Bay State Bldg., OALR

LYNN

JONES, E. W.....44 Atlanta St., Op.A
O'REILLY, W. F.....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F.....68 Walnut St., OALR

NORTH ADAMS

THOMPSON, G. H.....18 Ashland St., OALR

NORTHAMPTON

COLLINS, J. D.....90 Main St., ALR
DOW, F. E.....4 West St., Op.

PALMER

MOORE, G. A.....Bank Bldg., OALR

SALEM

HENNESSY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....4 Chestnut St., OALR
CARLETON, RALPH.....10 Temple St., Op.
IRWIN, V. J.....389 Main St., OALR
IRWIN, V. J., JR.....274 Main St., OALR

TAUNTON

BAKER, H. B.....58 Main St., OALR

WESTFIELD

CLARK, F. T.....52 Broad St., OALR

WORCESTER

CAHILL, J. W.....390 Main St., OALR
CROSS, A. E.....Slater Bldg., Op.
ESTABROOK, CHARLES.....390 Main St., OALR
MESSIER, A. EUGENE.....Park Bldg., OALR
RICE, JOHN E.....390 Main St., OALR

MICHIGAN

ANN ARBOR

FURSTENBERG, ALBERT C.....330 S. State St., ALR
SLOCUM, GEORGE.....328 E. Huron St., Op.

BATTLE CREEK

COLVER, B. N.....182 Manchester St., OALR
FARNESWORTH, M. A.....146 Greenwood St., OALR
FRAZER, ROBERT H.....14 Greenwood Ave., ALR
HAUGHEY, WILFRID.....24 W. Main St.,OALR
SLEIGHT, R. D.....Post Bldg., OALR
STEGMAN, L. B.....Sanitarium, Op.
WENCKE, C. G.....16 Wentworth Ct., OALR

BAY CITY

BAKER, C. H.....Crapo Block, OALR

DETROIT

AMBERG, EMIL.....David Whitney Bldg., ALR
BEATTIE, ROBERT.....David Whitney Bldg.,OALR
BENTLEY, NEIL.....1161 David Whitney Bldg., OALR
BERNSTEIN, E. J.....Professional Bldg., OALR
CAMPBELL, D. A.....David Whitney Bldg., OALR
CAMPBELL, DON M.....Peter Smith Bldg., OALR
CONNOR, RAY.....1410 Stroh Bldg., OALR
DEFNET, WM. A.....62 Adams St., W., ALR
FOWLER, WILLIAM.....Empire Bldg., OALR
GOUX, R. S.....545 David Whitney Bldg., OALR
HARTZ, H. J.....27 E. Adams St., ALR
HEATH, PARKER.....David Whitney Bldg., Op.
MAIRE, L. E.....Park Bldg., Op.A
MCCLELLAND, CARL C.....David Whitney Bldg., OALR

MERCER, R. E.....David Whitney Bldg., LR
MINER, S. C.....1564 Woodward Ave., ALR
MORTON, JOHN B.....90 E. Warren Ave., OALR
ODELL, ANNA.....32 Adams St., Op.AL
PARKER, WALTER R.....David Whitney Bldg., Op.
PEIRCE, HORACE W.....David Whitney Bldg., ALR
RENAUD, G. L.....Fine Arts Bldg., OALR
RYERSON, FRANK L.....Fine Arts Bldg., OALR
SANDERSON, H. H.....David Whitney Bldg., OALR
SHURLEY, B. R.....62 W. Adams St., ALR
SIMPSON, H. L.....David Whitney Bldg., ALR
WALKER, C. C.....Woodward at Warren, Op.
WENDEL, JACOB S.....David Whitney Bldg., ALR

ESCANABA

BOYCE, WILLIAM B.....1019 Ludington St., OALR

GRAND RAPIDS

DEAN, ALFRED.....304 Gilbert Blk. Bldg., Op.
GRANT, P. T.....Widdicomb Bldg., OALR
ROBERTSON, F. D.....Ashton Bldg., OALR
ROLLER, L. A.....122 Monroe St., OALR
WELSH, D. E.....Powers Theater Bldg., OALR

IONIA

SEDGWICK, O. W.....Box 494, OALR

IRON MOUNTAIN

BOYCE, G. H.....408 E. D. St., OALR

JACKSON

WINTER, G. E.....44 Sun Bldg., OALR

KALAMAZOO

FULKERSON, C. B.....Kalamazoo National Bank Bldg., OALR
GRANT, F. E.....Kalamazoo National Bank Bldg., OALR
WILBUR, E. P.....Kalamazoo National Bank Bldg., OALR

LANSING

BURDICK, AUSTIN F.....416 Tussing Blk., OALR

MENOMINEE

ELWOOD, CALVIN R.....Spies Bldg., OALR

MT. CLEMENS

CROMAN, JOSEPH M. JR.....Washington Apts., OALR

ST. JOSEPH

MCDERMOTT, JOHN J.....St. Joseph Sanitarium, OALR

SAGINAW

MCKINNEY, ALEXANDER R.....Bearinger Bldg., OALR
ROGERS, A. S.....105 S. Jefferson Ave., OALR
WATSON, ROY S.....120 N. Michigan Ave., OALR

TRAVERSE CITY

HOLDSWORTH, FRANK.....206 Wilhelm Blk., OALR

MINNESOTA

DULUTH

BRIGGS, F. W.....Lyceum Bldg., ALR
COLLINS, HOMER.....New Jersey Bldg., OALR
KNAPP, FRANK N.....203 W. Superior St., OALR
SHASTID, T. H.....Lyceum Bldg., Op.
TILDERQUIST, D. L.....7 E. Superior St., OALR
TURNBULL, F. M.....Providence Bldg., OALR
WINTER, JOHN A.....Fidelity Bldg., OALR

FERGUS FALLS

KITTLESON, THEODORE N.....101 Lincoln Ave., OALR

HIBBING

MORSMAN, L. W.....Congdon Bldg., OALR

MANKATO

JAMES, J. H.....National City Bank Bldg., OALR

MINNEAPOLIS

BEAUDOUX, H. A.....La Salle Bldg., Op.A
BISHOP, C. W.....La Salle Bldg., OALR
BROWN, E. J.....900 Nicollet Ave., OALR
CAMP, W. E.....Donaldson Bldg., OALR
CAMPBELL, RORT. A.....La Salle Bldg., OALR
CLARK, H. S.....Syndicate Blk., OALR
DORGE, RICHARD I.....76 Seymour Ave., S. E., OALR
HANSEN, E. W.....Donaldson Bldg., OALR
LEE, J. W.....804 Besse Bldg., OALR
LEWIS, J. D.....La Salle Bldg., OALR
LITCHFIELD, J. T.....Yeats Bldg., OALR
LOOMIS, E. A.....Donaldson Bldg., OALR
MACINE, J. S.....Donaldson Bldg., OALR
MATTHEWS, JUSTUS.....Metropolitan Bldg., LR
MORSE, J. A.....Donaldson Bldg., OALR
MORTON, HOWARD MCI.....Metropolitan Bk. Bldg., OALR
MURRAY, WM. R.....Nicollet Clinic, OALR
NEWHART, HORACE.....Donaldson Bldg., OALR
OBERG, C. M.....Syndicate Blk., OALR
PARKER, E. H.....La Salle Bldg., LR
PATTERSON, W. E.....La Salle Bldg., OALR
PHELPS, K. A.....La Salle Bldg., OALR
PRATT, F. J., JR.....Metropolitan Bldg., OALR
PRATT, J. A.....Metropolitan Bldg., OALR
REYNOLDS, J. S.....La Salle Bldg., OALR
SMITH, A. E.....Donaldson Bldg., OALR
STROUT, E. S.....74 S. 11th St., OALR
STROUT, G. E.....Donaldson Bldg., OALR
WATSON, J. A.....Phys. and Surg. Bldg., OALR
WOOD, DOUGLAS.....Donaldson Bldg., OALR
WRIGHT, C. D.....Metropolitan Bldg., Op.A

ROCHESTER

BENEDICT, W. L.....Mayo Clinic, Op.
HEMPSTEAD, BERT E.....Mayo Clinic, ALR
KNIGHT, MARY S.....Mayo Clinic, Op.
LILLIE, H. I.....Mayo Clinic, ALR
NEW, G. B.....Mayo Clinic, LR
PRANGEN, A. DE H.....Mayo Clinic, Op.

ST. PAUL

BERRISFORD, PAUL D.....Lowry Bldg., OALR
BINGER, H. E.....Lowry Bldg., OALR
BOECKMAN, EGIL.....Lowry Bldg., OALR
BRAY, E. R.....1440 Lincoln Ave., OALR
BROWN, JOHN C.....Lowry Bldg., OALR
BURCH, F. E.....Hamm Bldg., Op.
CONNOR, CHAS. E.....Hamm Bldg., OALR
FOGARTY, CHAS. W.....1826 Summit Ave., OALR
FULTON, JOHN E.....728 Lowry Bldg., OALR
HOWARD, W. H.....137 N. Fairview Ave., OALR
LARSEN, CARL E.....Lowry Bldg., OALR
LEWIS, WILLIAM W.....836 Lowry Bldg., OALR
MALONEY, T. J.....Lowry Bldg., OALR
NELSON, L. A.....Lowry Arcade, Op.A
ROTHSCHILD, H. J.....Lowry Bldg., OALR
SHELLMAN, J. L.....Lowry Bldg., OALR
WHEELER, MERRITT W.....Lowry Bldg., OALR
WOLD, KARL C.....7th and Wacouta Sts., OALR

MISSISSIPPI

MERIDIAN

GUTHRIE, J. M.....Rosenbaum Bldg., OALR

OXFORD

GUYTON, B. S.....Carter Bldg., OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., OALR
EDWARDS, C. J.....1422 Wash., OALR
HOWARD, E. F.....First National Bank Bldg., ALR

MISSOURI

AURORA

O'DELL, TIMOTHY.....112 A Madison Ave., OALR

CARTHAGE

POST, WINFRED B.....1380 S. Main St., OALR
POWERS, EVERETT.....Central National Bank Bldg., OALR

COLUMBIA

NOYES, G. L.....311 Hitt St., OALR
SNEED, C. M.....506 W. Broadway, OALR

KANSAS CITY

ALLEN, CHAS. E.....Waldheim Bldg., OALR
ALTRINGER, ARTHUR N.....Rialto Bldg., ALR
BEIL, J. W.....Argyle Bldg., OALR
BELLOWS, GEORGE E.....Rialto Bldg., Op.
BLAKESLEY, T. S.....Lathrop Bldg., OALR
CURDY, R. J.....Rialto Bldg., Op.
CURRAN, EDWIN J.....Waldheim Bldg., Op.
DAVIS, HARRY B.....Lathrop Bldg., OALR
FOSTER, HAL.....Chambers Bldg., ALR
HOWARD, J. W.....Rialto Bldg., OALR
KEITH, W. E.....Waldheim Bldg., OALR
KIMBERLIN, J. W.....Rialto Bldg., Op.
LANING, J. H.....Lathrop Bldg., ALR
LEA, J. A.....Rialto Bldg., ALR
LEMOINE, A. N.....Rialto Bldg., Op.
LEONARD, A. C.....Bryant Bldg., OALR
LICHTENBERG, J. S.....Rialto Bldg., Op.
LORIE, ALVIN.....Commerce Bldg., ALR
LUX, PAUL.....Commerce Bldg., ALR
MAY, JAMES W.....800 Minnesota Ave., Op.
MILLER, HUGH.....Chambers Bldg., OALR
MOSS, H. E.....Rialto Bldg., OALR
MOTT, J. S.....Argyle Bldg., OALR
MYERS, J. L.....Shukert Bldg., OALR
MCALESTER, A. W. JR.....Bryant Bldg., Op.
MCCORTY, V. W.....Rialto Bldg., ALR
PAINTER, A. M.....Sharp Bldg., ALR
PATTERSON, J. M.....Bryant Bldg., OALR
ROBERTS, S. E.....Federal Reserve Bank Bldg., ALR
RUSSELL, E. L.....Argyle Bldg., OALR
SCHUTZ, W. H.....Bryant Bldg., OALR
SELLERS, L. M.....Argyle Bldg., ALR
SHOEMAKER, S. A
U. S. Vet. Hosp., No. 67, 11th and Harrison Sts., OALR
SHUMATE, D. L.....Shukert Bldg., OALR
THOMASON, H. E.....Rialto Bldg., OALR
TUREMAN, H. G.....Rialto Bldg., OALR
YAZEL, H. E.....Grand Ave., Temple, OALR

MOBERLY

DUTTON, C. K.....OALR

ST. JOSEPH

FORGRAVE, L. ROBERT.....Logan Bldg., OALR
KENNEY, W. L.....Corby Forsee Bldg., OALR
MINTON, W. H.....Bartlett Bldg., OALR
PROUD, W. C.....Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M.....3858 Westminster Place, ALR
ARBUCKLE, M. F.....University Club Bldg., ALR

BALL, J. MOORES.....4500 Olive St., Op.
BARCK, CARL.....Humboldt Bldg., Op.A
BARDENHEIER, F. G. A.....Frisco Bldg., Op.A
BLACK, W. D.....Metropolitan Bldg., OALR
BRYANT, W. M. C.....University Club Bldg., ALR
BURNS, S. S.....Humboldt Bldg., ALR
CHARLES, J. W.....Humboldt Bldg., Op.
COSTEN, J. B.....Lister Bldg., ALR
DONNELL, NEWMAN R.....Metropolitan Bldg., Op.
DYER, CLYDE P.....Metropolitan Bldg., Op.
EWING, ARTHUR E.....Metropolitan Bldg., Op.
GOLDSTEIN, M. A.....3858 Westminster Pl., ALR
GREEN, JOHN JR.....Metropolitan Bldg., Op.
GUGGENHEIM, L. K.....Carleton Bldg., ALR
GUNDELACH, C. A.....825 University Club Bldg., ALR
HARDESTY, J. F.....Humboldt Bldg., Op.
HARDY, W. F.....Metropolitan Bldg., Op.
HIGBEE, E. H.....Metropolitan Bldg., Op.
HOURN, G. E.....University Club Bldg., ALR
JACOBS, M. W.....Carleton Bldg., Op.
JENNINGS, J. E.....Carleton Bldg., OALR
LAMB, HARVEY.....Metropolitan Bldg., Op.
LEAVY, C. A.....Metropolitan Bldg., ALR
LOEB, H. W.....Humboldt Bldg., ALR
LUEDDE, W. H.....Metropolitan Bldg., Op.
LYMAN, HARRY W.....Carleton Bldg., ALR
PFINGSTEN, C. F.....508 N. Grand Ave., ALR
PAYNE, RICHARD J.....University Club Bldg., ALR
POST, LAWRENCE.....Metropolitan Bldg., Op.
POST, MARTIN H.....Metropolitan Bldg., Op.
PROETZ, ARTHUR W.....University Club Bldg., ALR
REIM, HUGO.....Metropolitan Bldg., Op.
SAUER, W. E.....Humboldt Bldg., ALR
SCHOLZ, R. P.....Metropolitan Bldg., ALR
SCHWARTZ, F. A.....Metropolitan Bldg., Op.
SHAHAN, W. E.....Metropolitan Bldg., Op.
SHOEMAKER, W. A.....Carleton Bldg., Op.
SLUDER, GREENFIELD.....3542 Washington Ave., ALR
TOOKER, C. W.....Carleton Bldg., Op.
WESTLAKE, S. V.....Humboldt Bldg., ALR
WIENER, MEYER.....Carleton Bldg., Op.
WOODRUFF, F. E.....Metropolitan Bldg., Op.

SEDALIA

LOVE, J. G.....Ilgenfritz Bldg., OALR
TITSWORTH, GUY.....111 W. 4th St., Op.

SPRINGFIELD

BAILEY, H.....840 Landers Bldg., OALR
COFFELT, THEODORE A.....Woodruff Bldg., OALR
KLINGER, THOS. O.....Landers Bldg., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G.....Electric Light Bldg., OALR

BUTTE

DAYTON, G. O.....Lewisohn Bldg., OALR
DONAVAN, J. A.....Phoenix Bldg., OALR
MORSE, A. W.....Phoenix Bldg., OALR
POTTER, PETER.....Granate and Alaska Sts., OALR

GREAT FALLS

COULTER, C. F.....First Natl. Bank Bldg., OALR

LEWISTOWN

DAVIS, FRANK C.....ALR
PARSONS, J. G.....OALR

ROUNDUP

LEWIS, G. A.....29 Main St., OALR

NEBRASKA

BEATRICE

BAIRD, CHARLES G.....OALR
TUCKER, J. C.....OALR

COLUMBUS

CAMPBELL, C. H.....Columbus State Bank Bldg., OALR

KEARNEY

FOX, CHAS. H.....2116 Central Ave., OALR

LINCOLN

BROOKS, E. B.....Terminal Bldg., OALR
COOK, S. E.....Richards Block, OALR
CURTIS, W. L.....612 Security Mutual Bank Bldg., OALR
DAYTON, W. L.....Funke Bldg., OALR
FURGASON, A. P.....Richards Block., OALR
HOMPES, J. J.....Security Mutual Bldg., OALR
SANDERSON, DAVID D.....Funke Bldg., OALR
WILLIAMS, J. P.....Funke Bldg., OALR
WOODWARD, J. M.....Richards Block
ZEMER, S. G.....Security Mutual Bldg., OALR

NORFOLK

GADBOIS, A. E.....421 Norfolk Ave., OALR
NELSON, LLOYD L.....OALR

OMAHA

BANISTER, J. M.....Bankers Reserve Life Bldg., OALR
CALLFAS, W. F.....Brandeis Bldg., ALR
FAIRCHILD, NORA M.....600 Brandeis Theater Bldg., Op.
GIFFORD, HAROLD.....Brandeis Bldg., Op.A
GIFFORD, SANFORD R.....702 Brandeis Theater Bldg., Op.

HANEY, W. P.....Brandeis Theater Bldg., OALR
ISAAC, DAVID.....Brandeis Theater Bldg., OALR
KNOVDE, A. R.....Omaha National Bank Bldg., ALR
KULLY, B. M.....631 City Natl. Bank Bldg., ALR
LEMERE, H. B.....Bankers Reserve Life Bldg., OALR
OWEN, F. S.....Brandeis Bldg., OALR
PATTON, J. M.....Brandeis Bldg., Op.
POTTER, GEORGE B.....402 City Natl. Bank Bldg., OALR
POTTS, J. B.....Brandeis Bldg., ALR
RUBENDALL, CLARENCE.....Brandeis Bldg., OALR
TUCKER, J. C.....Brandeis Bldg., OALR
UREN, C. T.....Omaha National Bank Bldg., OALR
WHERRY, W. P.....Bankers Reserve Bldg., OALR

NEW HAMPSHIRE

NASHUA

NUTTER, C. F.....16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

FISHER, JAMES A.....Kinmouth Bldg., OALR
UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. C.....124 S. Illinois St., ALR
MCGIVERN, CHAS. S.....805 Pacific Ave., ALR
STICKNEY, O. D.....92 Pacific Ave., ALR

BLOOMFIELD

MOORE, JOHN D.....6 Washington St., OALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

JERSEY CITY

CHAMBERS, T. R.....15 Exchange Pl., OALR
PYLE, WALLACE.....18th and Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

CHATTIN, J. F.....671 Broad St., Op.
HUGHES, LEE W.....1019 Broad St., OALR
HURFF, J. W.....86 Washington St., OALR
KEIM, WILLIAM F.....2 Lombardy St., OALR
O'CONNOR, F. O.....671 Broad St., OALR
ORTON, H. B.....671 Broad St., ALR
QUINBY, W. O'G.....14 James St., OALR
SHERMAN, E. S.....Wiss Bldg., OALR
ZEHNDER, A. CHARLES.....180 Fairmount Ave., Op.

ORANGE

EMERSON, LINN.....Metropolitan Bldg., OALR

RIVERSIDE

SCHURTER, M. A.....8 Morton Ave., OALR

TRENTON

ADAMS, CHARLES.....52 W. State St., OALR
CRANE, J. WELLINGTON.....128 Third St., ALR

WEST HOBOKEN

SACCO, A. G.....440 Clinton Ave., OALR

NEW MEXICO

ALBUQUERQUE

BREHEMER, HARRISON L.....521 Luna Blvd., OALR

NEW YORK

ALBANY

BEDELL, A. J.....344 State St., Op.A
BEGGS, W. F.....2 Lombardy St., OALR
DOWLING, J. I.....116 Washington Ave., OALR
HINMAN, EUGENE E.....146 Washington Ave., ALR
JUDGE, H. V.....46 Willett St., Op.

BINGHAMTON

WATSON, H. D.....151 Front St., OALR

BROOKLYN

ALBRIGHT, LT. P. M....M. C. U. Navy, U. S. Naval Hosp., ALR
ARROWSMITH, HERBERT.....170 Clinton St., ALR
EVANS, JOHN N.....23 Schermerhorn St., Op.
GOLDING, JOSEPH E.....170 Hancock St., Op.
HANCOCK, J. C.....135 Cambridge Pl., Op.
KRUSKAL, ISAAC D.....863 Prospect Pl., Op.
LLOYD, RALPH I.....14 Eighth Ave., Op.
LOMBARDO, MELCHIORE.....186 Meserole St., Op.
MCCLELLAND, L. A.....78 McDonough St., ALR
MYERSON, MERVIN C.....184 Clinton St., ALR

BUFFALO

ANDREWS, H. D.....23 Allen St., Op.
BENNETT, A. G.....26 Allen St., Op.
BEYER, LOUIS J.....449 Delaware Ave., ALR
BLAAUW, E. E.....190 Ashland Ave., OALR
BOZER, HERRMANN E.....438 Delaware Ave., ALR
BROWN, C. M.....510 Delaware Ave., ALR
COTT, C. C.....483 Delaware Ave., ALR
COTT, DELZON N.....1001 Main St., ALR
COWPER, H. W.....543 Franklin St., Op.
EDSON, RAY.....560 Delaware Ave., Op.
FAIRBAIRN, JOHN.....503 Delaware Ave., ALR

FLAGG, J. D.....473 Virginia Ave., Op.A
FRANCIS, L. M.....636 Delaware Ave., Op.
GLOSSER, HERBERT H.....448 Franklin St., OALR
HEALEY, J. F.....503 Delaware Ave., ALR
HOWE, LUCIEN.....520 Delaware Ave., Op.
HUBBARD, A. E.....372 Franklin St., Op.
LEWIS, F. P.....454 Franklin St., Op.
MARCH, CLARA A.....465 Ashland Ave., Op.
PHILLIPS, W. L.....759 Richmond Ave., Op.
RENNER, W. S.....341 Linwood Ave., ALR
SATTERLEE, R. H.....187 Delaware Ave., Op.
SERNOFFSKY, I.....37 Allen St., Op.
STARR, E. G.....523 Delaware Ave., Op.
WEED, HARRY M.....196 Lincoln Ave., Op.

CORTLAND

HIGGINS, REUBEN P.....20 Court St., OALR

DUNKIRK

JACKLE, A. F.....63 E. 4th St., Op.

GENEVA

SPENGLER, J. A.....423 Main St., OALR

GLENS FALL

FIELDING, FRED G.....72 Ridge St., ALR

ITHACA

BULL, E. L.....124 E. State St., OALR
KIRKENDELL, J. S.....315 N. Aurora, OALR

JAMESTOWN

HOTCHKISS, W. W.....195 Forest Ave., OALR
REGER, H. S.....New Wellman Bldg., OALR

KENMORE

WURTZ, W. J. M.....2808 Delaware Ave., ALR

LIBERTY

DWORETSKY, JULIUS.....7 Law St., ALR

LOCKPORT

RINGUEBERG, EUGENE.....15 Main St., Op.

MT. VERNON

THOMSON, J. J.....3 Park Ave., OALR

NEW YORK CITY

ALMOUR, RALPH.....51 W. 73rd St., ALR
AUERBACH, JULIUS.....120 W. 86th St., ALR
BALLIN, M. J.....108 E. 60th St., ALR
BELL, GEO. HUSTON.....40 E. 41st St., Op
BERENS, CONRAD, JR.....30 E. 40th St., Op.
BOONE, JAMES I.....N. Y. Eye and Ear Infir., ALR
BRUDER, JOSEPH.....229 W. 79th St., OALR

CARTER, W. W.....24 E. 48th St., ALR
COHEN, MARTIN.....1 W. 85th St., OALR
COHN, FELIX.....31 E. 60th St., ALR
DAVIS, A. E.....47 E. 57th St., Op
DAVIS, G. E.....42 W. 77th St., OALR
DIXON, G. S.....40 E. 41st St., R
DUANE, ALEXANDER.....139 E. 37th St., Op.
DUNNINGTON, JOHN H.....104 E. 40th St., Op.
FORBES, H. H.....40 E. 41st St., ALR
FREUDENTHAL, WOLF.....24 W. 88th St., ALR
FRIDENBERG, P. H.....38 W. 59th St., OALR
FRIEDMAN, DAVID.....44 W. 77th St., ALR
GATEWOOD, W. L.....15 E. 48th St., ALR
GLOGAU, OTTO.....64 E. 91st St., OALR
GOLDMAN, HARRY G.....57 W. 58th St., ALR
GOTTLIEB, M. J.....814 West End Ave., ALR
GRUSHLOW, ISRAEL.....271 Central Park, W., ALR
GUTTMAN, J.....987 Madison Ave., OALR
HASKINS, W. H.....40 E. 41st St., ALR
HAYS, H. N.....22 W. 74th St., ALR
HETRICK, LLEWELLYN E.....20 E. 53rd St., ALR
HOPKINS, W. E.....515 Park Ave., OALR
HURD, L. M.....39 E. 50th St., ALR
IMPERATORI, C. J.....17 E. 38th St., ALR
INGERMAN, SERGIUS.....418 Central Park, W., OALR
IRWIN, F. N.....114 E. 54th St., OALR
JACOBS, S. M.....1018 E. 163rd St., OALR
JARECKY, HERMAN.....138 W. 86th St., OALR
JOHNSON, THOMAS H.....30 W. 59th St., Op.
JUDD, H. B.....47 E. 57th St., ALR
KERRISON, P. D.....58 W. 56th St., ALR
KEY, B. W.....7 W. 49th St., Op.
KING, J. J.....40 E. 41st St., ALR
KIRBY, DANIEL B.....30 W. 59th St., Op.
KNAPP, ARNOLD.....10 E. 54th St., Op.
KOPETZKY, S. J.....51 W. 73rd St., ALR
KRUG, E. F.....12 W. 44th St., OALR
LA VIGNE, A. A.....11 E. 48th St., OALR
LUBMAN, MAX.....135 E. 65th St., ALR
MAY, C. H.....698 Madison Ave., Op.
MAYBAUM, J. L.....12 E. 86th St., ALR
MAYER, EMIL.....40 E. 41st St., ALR
MCCOY, JOHN.....730 Fifth Ave., ALR
MEIERHOF, E. L.....1140 Madison Ave., OALR
MITTENDORF, A. D.....399 Park Ave., Op.
MITTENDORF, W. K.....115 E. 53rd St., Op.A
NISSELSON, MAX.....616 Madison Ave., OALR
OPPENHEIMER, SEYMOUR.....45 E. 60th St., OALR
PHILLIPS, W. C.....40 W. 47th St., ALR
REESE, R. G.....50 W. 52nd St., Op.
SCHILLER, A. N.....225 West End Ave., ALR

SCHLIVEK, KAUFMANN.....1000 Park Ave., Op.
SOMBERG, JOSEPH S.....45 E. 60th St., Op.
STARK, JESSE B.....270 Park Ave., Op.
STURGIS, LEIGH F.....128 W. 87th St., LR
VOORHEES, IRVING W.....14 Central Park, W., ALR
WEEKS, JOHN E.....20 E. 53rd St., Op.
WEEKS, WEBB W.....20 E. 53rd St., Op.
WHEELER, J. M.....30 W. 59th St., Op.
WIENER, ALFRED.....550 Park Ave., OALR
YANKAUER, SIDNEY.....616 Madison Ave., ALR

NIAGARA FALLS

PRICE, N. W.....445 Third St., OALR
ROOKER, A. L.....1347 Michigan Ave., ALR

NYACK-ON-HUDSON

TOMS, S. W. S..... ALR

OLEAN

TINDOLPH, LEA W.....First Natl. Bank Bldg., OALR

ONEIDA

CROCKETT, R. L..... Op.

POUGHKEEPSIE

KRIEGER, W. A.....36 Market St., OALR

ROCHESTER

BARBER, FRANK.....75 S. Fitzhugh St., Op.
CARROLL, G. G.....614 Main St., W., OALR
CLARK, L. H.....337 Monroe Ave., OALR
JONES, LEONARD W.....53 S. Fitzhugh St., OALR
LERNER, MACY L.....332 Park Ave., Op.
McDOWELL, N. D.....275 Alexander St., OALR
MORRIS, A. G.....41 Gibbs St., OALR
SHAPERO, I. M.....365 East Ave., OALR
SNELL, A. C.....53 S. Fitzhugh St., Op.

SYRACUSE

BROWN, MORTIMER G.....802 University Bldg., ALR
BRITTEN, G. S.....University Blk., OALR
BRUST, H. O.....720 S. Crouse Ave., ALR
FOWLER, S. R.....University Bldg., ALR
GILLETTE, DAVID F.....811 Park St., Op.
HALL, EDW. P..... OALR
KAUFFMAN, ARNOLD B.....Gurney Bldg., ALR
KLINE, H. G.....University Blk., ALR
MARLOW, SEARLE B.....University Bldg., Op.

TROY

SULZMAN, F. M.....1831 Fifth Ave., OALR

UTICA

BEATTIE, W. HENRY.....252 Genesee St., OALR

WATERTOWN

ATKINSON, WALTER S.....168 Sterling St., Op.

NORTH CAROLINA

ASHEVILLE

BRIGGS, H. H.....73 Haywood St., OALR
GREENE, JOSEPH E.....Haywood Bldg., ALR

CHARLOTTE

MATHESON, J. P.....511 Independence Bldg., OALR
PEELER, C. N.....W. 7th St., OALR
SLOAN, HENRY L.....Independence Bldg., Op.
WHISNANT, A. M.....Realty Bldg., OALR

GREENSBORO

REAVES, W. P.....117 W. Sycamore St., OALR

NEW BERN

DANIELS, R. L.....Elks' Temple, OALR

RALEIGH

WRIGHT, J. B.....Citizens Natl. Bank Bldg., OALR

SALISBURY

BRAWLEY, R. V.....Wallace Bldg., OALR

WILMINGTON

MURPHY, J. G.....Murchison Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W.....O'Hanlon Bldg., OALR

NORTH DAKOTA

FARGO

RINDLAUB, ELIZABETH P.....De Lendrecie Blk., OALR
RINDLAUB, J. H.....De Lendrecie Blk., OALR
RINDLAUB, M. P., JR.....De Lendrecie Blk., OALR

MINOT

MCCANNELL, A. D.....Colcord Bldg., OALR

WAHPETON

LANCASTER, WILSON.....310 Dakota Ave., OALR
RICE, C. P.....Sterns Bldg., OALR

OHIO

AKRON

BROWN, L. E.....2nd Natl. Bank Bldg., OALR
MOORE, T. K.....Hamilton Bldg., OALR
STEVENSON, D. W.....165 E. Market St., OALR

ALLIANCE

KING, G. L.....437 E. Market St., OAR

BELLEFONTAINE

BUTLER, ROBERT H.....227 W. Columbus Ave., OALR
HARBERT, J. P.....135 N. Ulain St., OALR

CANTON

SCHILD, E. H.....9th and Cleveland, Op.A

CINCINNATI

BEYER, ARTHUR G.....19 Garfield Pl., OALR
GOODYEAR, HENRY M.....3029 Fairfield Ave., ALR
HANS, C. L.....19 W. 7th St., OALR
HARRIS, W. C.....Groton Bldg., ALR
HINNEN, G. A.....1343 Delta Ave., OALR
IGLAUER, SAMUEL.....Livingston Bldg., ALR
JONES, CHARLES C.....19 Garfield Pl., ALR
KING, C.....Union Central Bldg., Op.
KING, EDWARD.....605 Traction Bldg., ALR
LAMB, F. W.....7th and Vine Sts., OALR
MITHOEFER, WILLIAM.....19 Garfield Pl., ALR
MURPHY, W. E.....7th and Race Sts., OALR
PHINNEY, F. D.....22 W. 7th St., OALR
RAY, VICTOR.....19 W. 7th St., OALR
SIEGEL, F. X.....2700 Union Central Bldg., OALR
STANBERRY, HENRY.....Doctors Bldg., Op AR
STEWART, T. M.....Union Trust Bldg., OALR
STRICKER, LOUIS.....Groton Bldg., Op.
TANGEMAN, H. F.....Central Bldg., ALR
THOMPSON, E. H.....Vindonissa Bldg., 19 W. 7th St., OALR
URNER, M. H.....Union Central Life Bldg., OALR
VAIL, D. T.....24 E. 8th St., Op.
VAIL, H. H.....24 E. 8th St., ALR

CLEVELAND

ABBOTT, W. J.....8314 Euclid Ave., OALR
BRUNER, ABRAM B.....Guardian Bldg., Op.
BRUNER, W. E.....Guardian Bldg., Op.
BURKE, T. A.....Rose Bldg., OALR
COGAN, J. E.....Rose Bldg., OALR
CUTLER, FRANKLIN E.....1025 Schofield Bldg., ALR
FORSYTHE, S. T.....1011 Euclid Ave., OALR
INGERSOLL, J. M.....Osborn Bldg., ALR
KENDALL, M. R.....Osborn Bldg., ALR
LARGE, S. H.....2323 Prospect Ave., ALR
LENKER, J. N.....Osborn Bldg., ALR
McCALL, JULIUS W.....Osborn Bldg., ALR
McDONALD, C. L.....Ainsfield Bldg., OALR
METZ, R. B.....Guardian Bldg., Op.
METZENBAUM, MYRON.....Rose Bldg., ALR
MONSON, S. H.....Ainsfield Bldg., OALR
MUSSUN, WILLIAM G.....Osborn Bldg., ALR
NELSON, C. F.....Schofield Bldg., OALR
PITKIN, C. E.....Osborn Bldg., ALR

PRENDERGAST, D.....1110 Euclid Ave., OALR
QUITTNER, S. S.....3912 Prospect Ave., OALR
RIEGER, WALTER H.....Hanna Bldg., ALR
RUST, E. G.....Hanna Bldg., Op.A
SHACKLETON, W. E.....2323 Prospect Ave., Op.
SHIRAS, H. H.....Guardian Bldg., Op.
SHIREY, OSCAR M.....Osborn Bldg., ALR
SIMONDS, O. F.....Hanna Bldg., Op.
STOTTER, A. L.....1148 Euclid Ave., OALR
STOTTER, JAMES.....1148 Euclid Ave., OALR
TRIPP, I. C.....Rose Bldg., OALR
TUCKERMAN, W. C.....Osborn Bldg., Op.
TUCKERMAN, W. H.....Osborn Bldg., OALR
WAUGH, J. M.....Cleveland Clinic, Euclid Ave. at 93rd St., ALR
WOLFENSTEIN, LEO.....Rose Bldg., Op.

COLUMBUS

BEATTY, H. G.....150 E. Broad St., OALR
BROWN, J. E.....370 E. Town St., OALR
CLARK, I. G.....188 E. State St., OALR
HAUER, A. M.....327 E. State St., OALR
PROUT, A. W.....370 E. Town St., OALR
SILBERNAGEL, C. E.....283 E. State St., OALR
SULZER, G. A.....200 E. State St., OALR
THOMAS, F. W.....74 S. 5th St., OALR
TIMBERMAN, ANDREW.....525 City Bk. Bldg., OALR
WOLFE, A. C.....350 E. State St., ALR
WRIGHT, J. W.....Central National Bank Bldg., Op.

DAYTON

BINKLEY, R. S.....Fidelity Med. Bldg., OALR
BLACKBURN, W. J.....Reibold Bldg., OALR
DUTROW, H. V.....Fidelity Medical Bldg., OALR
FARMER, A. G.....Fidelity Medical Bldg., OALR
FOUTS, JOHN D.....Fidelity Medical Bldg., OALR
HARRIS, H. B.....Fidelity Medical Bldg., OALR
KILBOURNE, PERLEY H.....Fidelity Medical Bldg., OALR
MILLETTE, J. W.....Reibold Bldg., OALR

ELYRIA

GILL, GEORGE.....146 Middle Ave., OALR

IRONTON

DUNN, O. B.....Fourth and Railroad Sts.

LIMA

STUEBER, F. G.....Main and North Sts., OALR
STUEBER, PAUL J.....209 W. North St., OALR

LORAIN

MONOSMITH, O. B.....424 Broadway, OALR

MANSFIELD

GARBER, J. M.....48 Park Ave., OALR
WISE, R. C.....News Bldg., OALR

MARIETTA

SAUER, W. W.....Box 416, OALR

MARION

BRICKLEY, D. W.....196 W. Center St., OALR

MIDDLETOWN

WILLIAMS, WALTER H.....OALR

NEWARK

HATCH, C. B.....3 W. Church St., OALR

SALEM

HILL, A. J.....301 Citizens Natl. Bank Bldg., OALR

SANDUSKY

BLISS, C. B.....411 Columbus Ave., OALR

SPRINGFIELD

EASTON, J. C.....Fairbanks Bldg., OALR
HARTLEY, F. A.....Fairbanks Bldg., OALR
HOGUE, D. W.....Fairbanks Bldg., OALR
MINOR, C. L.....Fairbanks Bldg., OALR

STEUBENVILLE

GOURLEY, G. F.....Sinclair Bldg., OALR

TIFFIN

PORTER, E. H.....85 Madison St., OALR

TOLEDO

ALDER, F. W.....Toledo Medical Bldg., OALR
ALDERDYCE, W. W.....Nasby Bldg., OALR
DENMAN, IRA O.....Ohio Bldg., OALR
HOBART, BERTHA K.....Produce Exchange Bldg., OALR
KING, CHARLES R.....Spitzer Bldg., OALR
LUKENS, CHARLES.....218 Michigan Ave., OALR
O'BRIEN, C. S.....Colton Bldg., Op
SNYDER, W. H.....211 Ontario St., OALR
STEINFELD, A. L.....234 Michigan St., OALR

XENIA

MADDEN, REID.....Allen Bldg., OALR
SHIELDS, L.....Allen Bldg., ALR

YOUNGSTOWN

BEARD, HAROLD J.....Dollar Bldg., OALR
GIBSON, R. D.....Dollar Savings and Trust Bldg., OALR
HARTZELL, S. M.....Dollar Bank Bldg., OALR

ZANESVILLE

BARON, F. S.....Peoples Bank Bldg., OALR
CULBERTSON, L. R.....Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D.....Central Natl. Bank Bldg., OALR

GUTHRIE

BARKER, CHAS. B.....224½ W. Oklahoma Ave., OALR

MCALISTER

DAVIS, J. E.....Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M.....Barnes Bldg., OALR

OKALAHOMA CITY

DIXON, W. E.....706 1st Natl. Bank Bldg., OALR
FERGUSON, E. I.....607 1st Natl. Bank Bldg., OALR
GUTHRIE, A. L.....Amer. Natl. Bank Bldg., OALR
MCHENRY, D. D.....Med. Arts Bldg., OALR
NEWTON, L. A.....Med. Arts Bldg., OALR
TODD, H. C.....Colcord Bldg., OALR

TULSA

BRASWELL, C. J.....726 Mayo Bldg., OALR
COOK, W. ALBERT.....506 Palace Bldg., OALR
ROTH, A. W.....Palace Bldg., OALR

OREGON

LA GRANGE

BOUVY, HARRY.....New Foley Bldg., OALR

PORTLAND

AINSLIE, GEORGE.....Oregonian Bldg., OALR
BRUERE, G. E.....Journal Bldg., OALR
DAVIS, RALPH F.....Selling Bldg., OALR
FENTON, RALPH A.....616 Journal Bldg., OALR
HENTON, GEORGE EARL.....Morgan Bldg., OALR
JOHNSTON, WILSON.....Stevens Bldg., OALR
KIEHLE, F. A.....Corbett Bldg., OALR
KISTNER, F. B.....Stevens Bldg., ALR
MCCOOL, JOSEPH L.....Stevens Bldg., Op.

PENNSYLVANIA

ALTOONA

GLOVER, S. P.....1118 12th Ave., OALR

BEAVER

SCROGGS, J. J.....218 E. 3rd St., OALR

BETHLEHEM

CHASE, WALTER D.....230 E. Broad St., ALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L.....Reiberg Bldg., OALR
HAZLETT, L. R.....117 S. Main St., OALR

CHARLEROI

FERMAN, JOHN W.....Stahlman Bldg., OALR
STAHLMAN, F. C.....Stahlman Bldg., OALR

CHESTER

CROSS, G. H.....525 Welsh St., Op.

CYNWYD

TUCKER, GABRIEL E.....335 Trevor Lane, ALR

ERIE

DENNIS, D. N.....221 W. 9th St., Op A
DENNIS, E. P.....311 W. 10th St., OALR
DUNN, I. J.....Masonic Temple, OALR
FUST, J. H.....138 W. 9th St., OALR
HEARD, C. F.....Masonic Temple, OALR
JACKSON, J. D.....2324 W. 8th St.
RUSSELL, J. A.....206 Masonic Temple, ALR
SCHLINDWEIN, G. W.....138 W. 9th St., OALR
SHREVE, O. M.....162 W. 8th St., OALR
WRIGHT, KATHERINE L.....247 W. 8th St., ALR

FRANKLIN

JOBSON, G. B.....Printz Bldg., OALR

GREENSBURG

FAIRING, JOHN W.....204 Tribune Bldg., OALR
MCKEE, C. W.....Coulter Bldg., OALR

HARRISBURG

DAILY, G. I.....713 N. 3rd St., OALR
FARNSLER, H. H.....1438 Market St., OALR
GRACEY, GEORGE F.....209 State St., OALR
PARK, J. W.....32 N. 2d St., OALR
REBUCK, C. S.....412 N. 3d St., OALR

HAZLETON

REICHE, O. C.....328 W. Broad St., OALR

HOMESTEAD

BROWN, W. E.....8th Ave. and Amity St., OALR

HUNTINGDON

SEARS, W. H.....514 Penn St., OALR

JOHNSTOWN

BARKER, O. G. A.....Johnstown Trust Bldg., Op.
HARRIS, CLARENCE M.....Johnstown Trust Bldg., OALR
RUSH, CALVIN C.....342 Main St., OALR

MIDDLETOWN

GEORGE, H. W.....19 N. Union St., Op.

NEW KENSINGTON

MCCONNELL, T. E.....Alter Bldg., OALR
STEIM, JOSEPH M.....1st Natl. Bk. Bldg., Op.

NORTH EAST

HEARD, MARY K.....41 W. Main St., Op.

PHILADELPHIA

ALEXANDER, GEORGE J.....1831 Chestnut St., ALR
ANNON, WALTER T.....259 S. 17th St., ALR
APPLEMAN, L. F.....308 S. 16th St., Op.
BABBITT, JAMES A.....2117 Chestnut St., ALR
BALDWIN, KATE W.....1117 Spruce St., ALR
BALENTINE, P. L.....1524 Chestnut St., Op.
BOEHRINGER, H. W.....1811 S. 22nd St., Op.
BROWN, SAMUEL HORTON.....1901 Mt. Vernon St., Op.
BRUMM, SETH A.....Stock Exchange Bldg., ALR
BUTLER, MARGARET F.....1831 Chestnut St., ALR
BUTT, M. M.....2045 Chestnut St., Op.
CLERF, LOUIS H.....128 S. 10th St., ALR
COATES, G. M.....1736 Pine St., ALR
COHEN, SAMUEL.....1626 Spruce St., ALR
CRAMPTON, G. S.....1700 Walnut St., Op.
CREIGHTON, W. J.....1905 Chestnut St., Op.
CROSKEY, J. W.....Aldine Hotel, Op.
DAVIS, J. L.....135 S. 18th St., ALR
DEICHLER, LYN WALTER.....2028 Chestnut St., OALR
DINTENFASS, HENRY.....1714 Pine St., ALR
EARL, HARRY D.....3717 Spruce St., ALR
ERSNER, M. S.....1729 Pine St., ALR
FELT, C. L.....N. E. Cor. Chestnut & 20th Sts., ALR
FOX, L. W.....303 S. 17th St., Op.
GLEASON, E. B.....2033 Chestnut St., ALR
GODDARD, H. M.....1531 Spruce St., ALR
GRISCOM, J. M.....1925 Chestnut St., Op.
HAWMAN, E. G.....4051 Broad St., OALR
HEED, C. R.....1205 Spruce St., Op.
HERBERT, A. W.....119 E. Lehigh Ave., OALR
HOLLOWAY, T. B.....1819 Chestnut St., Op.
HUSIK, DAVID N.....1610 Spruce St., ALR
JACKSON, C.....128 S. 19th St., L
KAUFMAN, A. S.....1923 Spruce St., ALR
KEELER, J. C.....254 S. 16th St., ALR
KRAUSS, FREDERICK.....1701 Chestnut St., OALR
LAESSLE, H. A.....The Lenox, 13th and Spruce Sts., ALR
LAWRENCE, GRANVILLE A.....Medical Arts Bldg., Op.
LEWIS, FIELDING O.....259 S. 17th St., ALR
LOFTUS, J. E.....605 Medical Arts Bldg., ALR
LOVE, LOUIS F.....1207 Spruce St., Op.
LUKENS, R. McD.....1308 Hunting Park Ave., ALR
MACFARLAN, DOUGLAS.....1805 Chestnut St., OALR
MACKENZIE, ALICE V.....1724 Spruce St., OALR
MACKENZIE, GEORGE W.....1724 Spruce St., ALR
MILLER, E. B.....2008 Walnut St., Op.
MITCHELL, E. K.....704 Lehigh Ave., ALR

MOORE, W. F.....255 S. 16th St., ALR
OLSHO, SYDNEY L.....235 S. 15th St., OALR
O'REILLY, C. A.....1901 Chestnut St., ALR
PETER, LUTHER C.....N. E. Cor. Chestnut and 20th St., Op.
PONTIUS, P. J.....1831 Chestnut St., OALR
POSEY, WM. CAMPBELL.....21st and Chestnut Sts., Op.
RADCLIFFE, MCCLUNEY.....1906 Chestnut St., Op.
REESE, WARREN S.....230 S. 21st St., Op.
RIDPATH, R. F.....1928 Chestnut St., ALR
ROMMELL, J. C.....4501 N. Broad St., OALR
SARTAIN, PAUL J.....2006 Walnut St., Op.
SCARLETT, HUNTER W.....2228 Locust St., Op.
SCHATZ, H. A.....2035 Chestnut St., ALR
SCHNEIDEMAN, T. B.....Professional Bldg., Op
SCHWEINITZ, G. E. DE.....1705 Walnut St., Op.
SCHWENK, P. N. K.....1417 N. Broad St., Op.
SENER, W. J.....130 S. 18th St., OALR
SHEMELEY, W. G., JR.....1724 Spruce St., OALR
SKILLERN, R. H.....1928 Chestnut St., LR
SKILLERN, SAMUEL R., JR.....2008 Walnut St., ALR
SMITH, S. MCC.....1420 Spruce St., ALR
SPENCER, W.....1830 S. Rittenhouse Sq., OALR
STILES, C. M.....1831 Chestnut St., Op.
STOUT, PHILIP S.....Medical Arts Bldg., ALR
STROUSE, FRED M.....1301 Spruce St., ALR
SWEET, W. M.....1205 Spruce St., Op.
WAGERS, A. J.....The Lenox, 13th and Spruce Sts., ALR
WELCH, R. S. G.....League Island Nav. Hosp., OALR
WILLIAMS, H. J.....5908 Green St., Germantown, ALR
WHELAN, GEORGE L.....257 S. 16th St., ALR
ZENTMAYER, WILLIAM.....1506 Spruce St., Op.

PITTSBURGH

BEANE, GEORGE W.....Jenkins Arcade Bldg., OALR
BERNATZ, C. F.....Park Bldg., Op.
BLAIR, W. W.....Diamond Bank Bldg., Op.
CARSON, W. E.....Jenkins Arcade Bldg., Op.
CRAIGHEAD, NANCY B.....Jenkins Arcade Bldg.,
CURRY, G. E.....Westinghouse Bldg., Op.
DAY, EWING W.....Westinghouse Bldg., ALR
DICKINSON, B. M.....5634 Stanton Ave., E. Liberty Sta., ALR
FISCHER, NATHANIEL A.....Union Trust Bldg., ALR
FRANK, A. C.....Diamond Bank Bldg., ALR
GROSS, JULIUS E.....Jenkins Arcade Bldg., OALR
GUERINOT, A. J.....Jenkins Arcade Bldg., OALR
HAGEMAN, JOHN A.....Highland Bldg., ALR
HECKEL, E. B.....Jenkins Arcade Bldg., Op.
KNEEDLER, G. C.....Jenkins Arcade Bldg.,
KREBS, A.....Jenkins Arcade Bldg., Op.
LINN, J. G.....Highland Bldg., OALR

MARKEL, J. C.....Westinghouse Bldg., Op.
MARSHALL, WATSON.....Diamond Bank Bldg., ALR
MCCREADY, J. H.....Empire Bldg., ALR
MCKEE, GEORGE J.....1118 Westinghouse Bldg., ALR
MACLACHLAN, A. A.....May Bldg., ALR
MEANOR, W. C.....Westinghouse Bldg., Op.
MURDOCH, J. F.....Bessemer Bldg., Op.
PATTERSON, E. J.....Westinghouse Bldg., ALR
SEEGMAN, SIMON.....Jenkins Arcade Bldg., ALR
SHUMAN, G. H.....Park Bldg., Op.
SIMPSON, JOHN R.....1005 Westinghouse Bldg., ALR
SMITH, S. S.....Jenkins Arcade Bldg., Op.
STIEREN, EDWARD.....Union Trust Bldg., Op.
STIMSON, GEO. W.....Jenkins Arcade Bldg., ALR
STURM, S. A.....Jenkins Arcade Bldg., Op.
TAYLOR, MARTIN C.....415 Warrington Ave., Op.
TODD, G. C.....307 Westinghouse Bldg., ALR
TURNER, HUNTER H.....Jenkins Arcade Bldg., Op.
VAN KIRK, V. E.....Union Arcade Bldg., Op.
WEIMER, EDGAR S.....332 Lehigh Ave., Op.
WEISSER, E. A.....Empire Bldg., Op.
WIBLE, E. E.....Diamond Bank Bldg., Op.

READING

STOCKLER, J. A.....332 N. 5th St., OALR

RIDGEWAY

MCALISTER, J. C.....10 S. Mill Ave., OALR

SCHUYLKILL HAVEN

MOORE, GEORGE H.....200 Main St., OALR

SCRANTON

CORSER, J. B.....Scranton Private Hospital, OALR
CURTIN, EUGENE A.....Connell Bldg., OALR
FREY, C. L.....529 Vine St., OALR
SULLIVAN, JOHN J., JR.....Traders Bank Bldg., ALR

SHARON

NORTON, ROY R.....107 B. E. State St., OALR

SUNBURY

BECKER, H. M.....49 S. 4th St., OALR

UPPER DARBY

SHANNON, C. E.....104 Copley Rd., Op

WARREN

BALL, M. V.....214 Penn Ave., W., OALR

WASHINGTON

MCKENNAN, J. W.....Washington Trust Bldg., Op.
MCMURRAY, J. B.....Washington Trust Bldg., OALR

WILKES-BARRE

BUCKMAN, E. V.....70 S. Franklin St., OALR
CARR, G. W.....54 S. Franklin St., OALR
KISTLER, O. F.....43 N. Franklin St., Op.A
MCLAUGHLIN, P. A.....73 S. Washington St., OALR
SCHAFFERT, N. L.....57 S. Washington St., OALR
TAYLOR, LEWIS H.....83 S. Franklin St., OALR

YORK

KLINEDINST, J. F.....220 S. George St., OALR

RHODE ISLAND

NEWPORT

JACOBY, D. P. A.....106 Touro, Op.AL

PROVIDENCE

ADAMS, F. M.....224 Thayer St., ALR
CAPRON, F. P.....118 Angell St., OALR
HARVEY, N. D.....114 Waterman Ave., OALR
LEECH, J. W.....111 Broad St., OALR
MESSINGER, H. C.....170 Broad St., Op.
MUNCY, W. M.....25 Waterman St., OALR
PORTER, L. B.....195 Thayer St., OALR

SOUTH CAROLINA

CHARLESTON

KOLLOCK, C. W.....86 Wentworth, OALR
TOWNSEND, JOHN F....85 Wentworth, Cor. St. Phillips St., OALR

COLUMBIA

WHALEY, E. M.....1430 Blanding St., OALR

GREENVILLE

CARPENTER, E. W.....201 E. North St., ALR
MAULDIN, L. O.....Wallace Bldg., OALR

ROCK HILL

TWITTY, W. C.....Peoples Natl. Bank Bldg., OALR

SOUTH DAKOTA

ABERDEEN

ALWAY, ROBERT D.....423 S. Lincoln St., OALR

HURON

GROSVENOR, L. N.....378 Dakota St., OALR

MITCHELL

BOBB, E. V.....First Natl. Bank Bldg., OALR

RAPID CITY

WALSH, JOSEPH M..... OALR

SIOUX FALLS

GREGG, J. B.....Security Natl. Bank Bldg., OALR
PUTNAM, F. I.....708 Sioux Falls Natl. Bank Bldg., OALR

WATERTOWN

JOHNSON, A. E.....Citizens Bank Bldg., OALR

YANKTON

HOHF, JULIUS.....Clinic Bldg., OALR

TENNESSEE

CHATTANOOGA

HACKNEY, F. J.....630 Volunteer Bldg., OALR
HOGSHEAD, J. M.....602 Georgia Ave., OALR
JOHNSON, O. H.....630 Volunteer Bldg., OALR

DYERSBURG

BREWER, JOHN D.....618 Main St., OALR

JACKSON

DANCY, A. B.....First National Bank Bldg., OALR

KNOXVILLE

KINCAID, J. H.....Ross Flats, OALR
REAVES, CHAS. R.....Box No. 187, OALR

MEMPHIS

BLASSINGAME, CHARLES D.....Exchange Bldg., ALR
BLUE, J. B.....Exchange Bldg., OALR
ELLETT, E. C.....Exchange Bldg., Op.
FARRINGTON, P. M.....1473 Goodbar Ave., ALR
HARRIS, W. ROBIN.....Exchange Bldg., ALR
LEVY, LOUIS.....Bank of Commerce Bldg., OALR
LEWIS, A. C.....Exchange Bldg., OALR
MCKINNEY, R.....Bank of Commerce and Trust Bldg., ALR
MINOR, H. F.....Randolph Bldg., OALR
SHEA, J. J.....Exchange Bldg., OALR
SIMPSON, W. L.....Exchange Bldg., OALR
STANFORD, J. B.....Exchange Bldg., OALR

NASHVILLE

CAYCE, E. B.....Hitchcock Bldg., OALR
CULLOM, MARVIN MCT.....208 Hitchcock Bldg., OALR
HASTY, FREDERICK E.....Lambuth Bldg., OALR
KENNON, WILLIAM G.....Lambuth Bldg., OALR
WOOD, HILLIARD.....Independence Bldg., OALR

TEXAS

AUSTIN

BOERNER, MORRIS H.....Scarborough Bldg., OALR

BROWNWOOD

ANDERSON, W. B.....507 Main Ave., OALR

CUERO

DUCKWORTH, GUILFORD M.....Buchel Bldg., OALR

DALLAS

BETTISON, D. L.....502 Wilson Bldg., OALR
CARPENTER, E. R.....Medical Arts Bldg., OALR
CARY, E. H.....Southwestern Life Bldg., OALR
DECHERD, H. B.....Wilson Bldg., OALR
MCREYNOLDS, J. O.....Trust Bldg., OALR
NEWTON, FRANK H.....Mercantile Bank Bldg., OALR
SEAY, D. E.....Dallas Co. State Bank Bldg., OALR

EL PASO

IRVINE, E. H.....321 W. Rio Grande St., OALR
SCHUSTER, STEPHEN A.....1st Natl. Bank Bldg., ALR

FORT SAM HOUSTON

SHERMAN, MAJOR A. EDWARD.....Station Hospital, OALR

FORT WORTH

HOWARD, E. L.....First Natl. Bank Bldg., OALR
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HOUSTON

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HADEN, H. C.....913 Union Natl. Bank Bldg., OALR
ISRAEL, N. E.....Carter Bldg., OALR
ISRAEL, SIDNEY.....Carter Bldg., OALR
RALSTON, W. W.....1300 Walker Ave., Op.

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WOODSON, J. M.....City National Bank Bldg., OALR

TEXARKANA

FULLER, T. E.....Texarkana Natl. Bank Bldg., OALR
MANN, R. H. T.....State Natl. Bank Bldg., OALR

TYLER

VAUGHAN, E. H.....113½ W. Ferguson St., OALR

WACO

AYNSWORTH, HORACE.....Amicable Bldg., OALR

UTAH

OGDEN

HETZEL, C. C.....Eccles Bldg., OALR

PROVO

MERRILL, H. G.....35 East, 12 South St., OALR

SALT LAKE CITY

DONOHER, W. D.....Boston Bldg., OALR
HAMPTON, R. H.....Boston Bldg., OALR
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SNOW, LESLIE W.....Deseret Bank Bldg., OALR
STAUFFER, FREDERICK.....Deseret Bank Bldg., OALR

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RUTLAND

MARSHALL, GEORGE G.....Gryphon Bldg., OALR

VIRGINIA

RICHMOND

GATEWOOD, EMMETTE T.....Professional Bldg., ALR
HILL, EMORY.....Professional Bldg., Op.
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MILLER, C. M.....Stuart Circle Hospital, OALR

ROANOKE

GARRETT, J. R.....Strickland Bldg., OALR
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WASHINGTON

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BELLINGHAM

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EAU CLAIRE

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GREEN BAY

BELLIN, JOSEPH.....322 N. Washington St., OALR
COMEE, W. C.....Bellin Bldg., OALR
NADEAU, E. G.....Bellin-Buchanan Bldg., OALR

JANESVILLE

LINTLEMAN, F. R.....Hayes Blk., OALR

KENOSHA

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LA CROSSE

BRADFIELD J. A. L.....State Bank Bldg., OALR
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MADISON

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MANITOWOC

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OSHKOSH

ALLEN, L. P.....19 Jefferson Ave., OALR

RACINE

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WILLARD, L. M.....520 Third St., OALR

WYOMING

CHEYENNE

BECK, F. L.....408 Hynds Bldg., OALR
STRADER, G. L.....408 Hynds Bldg., OALR

AFRICA

NATAL

DURBAN

JOHNSON, LINDSAY.....Britannia Bldg.

CANADA

ALBERTA

EDMONTON

BROWN, W. H.....Bank of Montreal Bldg., OALR

BRITISH COLUMBIA

VANCOUVER

BOUCHER, R. B.....Banks Bldg., OALR

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KEYS, MORTON J.....307 Jones Block, OALR

MANITOBA

BRANDON

MCDIARMID, H. O.....Rossner Ave. and 9th St., OALR

NEW BRUNSWICK

ST. JOHN

CHIPMAN, LEVERETT DEV.....42 Coburg St., OALR

NOVA SCOTIA

HALIFAX

CUNNINGHAM, ALLAN R.....260 Barrington St., OALR

ONTARIO

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BUCHANAN, NORMAN D.....479 Water St., OALR

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GOLDSMITH, PERRY G.....84 Carlton St., ALR
HILL, C. E.....160 Bloor St., W., Op.
MACFARLANE, MURRAY.....190 Bloor St., OALR
MCCULLOUGH, R. J. P.....102 College St., OALR
MCKELVEY, A. D. M.....160 Bloor St., ALR
RISDON, ERNEST F.....30 Avenue Rd., ALR

QUEBEC

MONTREAL

BIRKETT, H. S.....252 Mountain St., ALR
BALLON, DAVID H.....255 Bishop St., ALR
BOUSQUET, J. P. E.....26 Sherbrook St., W., OALR
LASSALLE, ALBERT.....493 St. Denis St., OALR
MACMILLAN, JOHN A.....129 Stanley St., Op.
MCKEE, S. HANFORD.....158 Crescent St., Op.
ROGERS, J. T.....758 Sherbrook St., W., ALR
ROSENBAUM, J.....206 Bishop St., Op.
WHITE, E. H.....589 Dorchester St., ALR

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HAVANA

FERNANDEZ, FRANCISCO M.....Prado, 105, OALR

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PRAGUE

ELSCHNIG, ANTON.....Ferdinandstrasse, 10

ENGLAND

LONDON

ELLIOT, LT.-COL. R. H...54 Welbeck St., Cavendish Sq., W.
DUNDAS-GRANT.....148 Harley St., W.
KIRKPATRICK, LT.-COL. HENRY.....54 Welbeck St., W. 1.
LAWFORD, J. B.....27 Weymouth St., Portland Pl.
LISTER, SIR WILLIAM T.....24 Devonshire Pl., W. 1.

HOLLAND

LEIDEN

VAN DER HOEVE, PROF. J.....Rijnsburgerweg 6A.

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GLASGOW

GRAY, A. A.....

SWITZERLAND

ZURICH

BARKAN, A.....Credit Swisse, OALR
NAGER, PROF. F.....Freiestrasse 20

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